FIFTY TUTORIAL ESSAYS IN CLINICAL PSYCHOLOGY

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An independent academic psychologist, based in England, who has written extensively on different areas of psychology with an emphasis on the critical stance towards traditional ideas.

A complete listing of his writings at http://kmbpsychology.jottit.com.

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1. AUTOBIOGRAPHICAL MEMORY AND DEPRESSION

Williams and Broadbent (1986) were the first to note that individuals who attempted suicide by self-poisoning recalled autobiographical memories (AMs) in a different ways to matched controls. These individuals tended to recall general AMs rather than specific ones when asked to recall memories of events on particular days and in particular places. "It is suggested that overgeneral autobiographical memory is used to ward-off intrusive memories which occur during episodes of depression.." (Burnside et al 2004 p366).

The tendency to recall general AMs should also be more evident among individuals who have experienced childhood trauma. Henderson et al (2002) found that female students who admitted to experiencing childhood sexual abuse were more likely to report general AMs.

Burnside et al (2004) interviewed forty-one women who had previously reported childhood sexual abuse, defined as "any unwanted or coerced sexual contact by an adult or by another minor, where the victim clearly did not consent, which occurred before the age of sixteen" (p369). A measure of depression (Beck Depression Inventory; Beck et al 1974) was taken as well as an Autobiographical Memory Test (AMT) involving recall of specific events in their lives in response to cue cards (positive, negative or neutral words)(table 1.1). Non-specific (general) recall included general statements like "I used to walk the dog every day".

POSITIVE CUES	NEGATIVE CUES	NEUTRAL CUES
• Happy • Relieved • Eager • Sunny • Proud	• Ugly • Guilty • Failure • Worse • Hopeless	• Grass • Gigantic • Absence • Bread • Search

Table 1.1 - Memory cue words used by Burnside et al (2004).

The women were divided into two groups for analysis based on those who had experienced major depression (previous major depression; PMD)(n = 22) and those who had not (no previous major depression; NPMD)(n = 19). The PMD group recalled significantly less specific memories than the WPMD group generally (mean 2.11 vs 3.05), and significantly less in response to the negative cue words (mean 1.11 vs 2.11). There was no difference for the

positive cue words. Overall, individuals who had experienced depression did overgeneralise their AMs.

EVALUATION

There are a number of methodological issues related to this study.

1. The study was dependent on the memory of the women, and there was no independent verification of any of the events recalled including the childhood sexual abuse experienced.

2. The women's individual memory ability was a factor: "It is possible that women who have difficulty in recalling negative events are more likely to under-report episodes of depression and therefore be placed erroneously in the NPMD group" (Burnside et al 2004 p373).

3. Some of the findings appeared contradictory. For example, analysis of type of recall (general or specific) and aspects of the abuse. Women who had experienced abuse for longer and abuse that started at a younger age recalled more specific events. This appears to be contrary to the idea of overgeneral AMs as a defence against intrusive memories. But these relationships are not for the whole sample.

4. The study did not have a non-childhood sexual abuse control group for comparison.

5. To some degree it was assumed that type of AMs predicted depression, but it was possible that depression changed the type of AMs recalled. Depression is caused because specific memories are not assimilated properly, but during depression or recovery this process occurs (Williams et al 1999; assimilation model).

6. Burnside et al (2004) admitted that: "The results of this study represent a 'snapshot' of participants' memory styles in relation to their experiences so far. We do not know, for example, how many of the NMPD group may experience depression in the future" (p375). Thus the need for a longitudinal study.

7. Standardised measures were used in the study.

8. Because of the sensitive nature of the study, recruitment of the women who admitted to experiencing childhood sexual abuse is difficult. Burnside et al used fifty women who had taken part in a previous study (Hill

et al 2001) on childhood experiences and had reported the childhood sexual abuse then. Two women refused to take part in this study, and seven could no longer be contacted.

9. The AMT was administered first to avoid the participants realising the purpose of the study or being affected by the other measures.

10. The women were divided into two groups for analysis after the data had been collected. This runs the risk of groups of different sizes, or subjective decisions in order to make the groups equal size. It is better to have established the groups before data collection.

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2. CALLOUS AND UNEMOTIONAL TRAITS AND "FLEDGLING PSYCHOPATHS"

There is interest in successfully identifying "fledgling psychopaths" (Lynam 1996); ie: children who show persistent anti-social behaviour aNd as they grow up become fully-blown anti-social psychopaths. One attempt to identify these fledglings is by the presence of callous and unemotional traits (CUTs), which is what Moran et al (2008) investigated in a British sample (1).

The British Child and Adolescent Mental Health Survey 2004 approached a stratified sample (2) of 10 496 families, and 76% agreed to participate (3). Parents of children aged 5-16 years were interviewed face-to-face using the Strengths and Difficulties Questionnaire (SDQ) (Goodman et al 2000) (4). The child's nominated teacher was given a specially-designed questionnaire about CUTs (table 2.1).

ITEM	SCORED
1. Makes a good impression at first but people tend to see through him/her after they get to know him/her	0 = not true 1 = partly true 2 = certainly true
2. Shallow and fast-changing emotions	0 = not true 1 = partly true 2 = certainly true
3. Too full of his/her own abilities	0 = not true 1 = partly true 2 = certainly true
4. Is usually genuinely sorry if s/he has hurt someone or acted badly	0 = certainly true 1 = partly true 2 = not true
5. Can be seen as cold-blooded or callous	0 = not true 1 = partly true 2 = certainly true
6. Keeps promises	0 = certainly true 1 = partly true 2 = not true
7. Genuine in his/her expression of emotions	0 = certainly true 1 = partly true 2 = not true

(Range 0-14 with higher score = more CUTs)

(After Moran et al 2008)

Table 2.1 - Specially-designed questionnaire for teachers to measure CUTs.

At twelve and twenty-four months, parents were

posted a follow-up SDQ to complete. This longitudinal element of the study allowed the researchers to test a predictive model (figure 2.1).

TEACHER RATING OF CUTs AT BASELINE ⇒ PARENT RATING OF BEHAVIOUR PROBLEMS ON SDQ 12 AND 24 MONTHS LATER

Figure 2.1 - Predictive element of study.

Data were collected from 5770 children's teachers, and on 4609 children at follow-up.

The mean score for the teachers' questionnaire was 1.65 (out of 14). Higher scores were associated with male, older, black and minority ethnicity, poorer health, parental mental illness, large family size, and lower household income.

At follow-up a number of patterns were significant:

- CUTs predicted conduct disorder symptoms in boys in particular (figure 2.2);
- CUTs predicted high SDQ scores at follow-up, especially in boys;
- Behaviour problems at follow-up were associated with lower household income, parental mental illness, and high SDQ score at baseline (figure 2.3).
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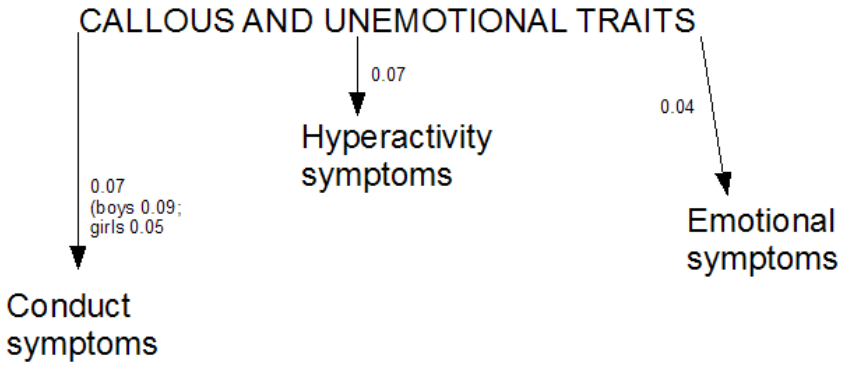
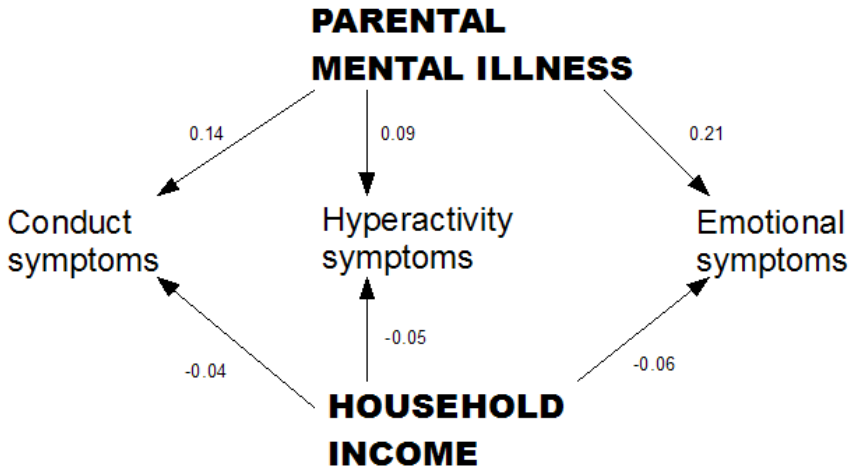


(All significant at p<0.001, except girls p<0.05)

Figure 2.2 - Correlations between CUTs and problem behaviour.



(All significant at p<0.001)

Figure 2.3 - Correlations between parental mental illness and household income, and problem behaviour.

CUTs was one of a number of factors associated with later anti-social behaviour (figure 2.4).

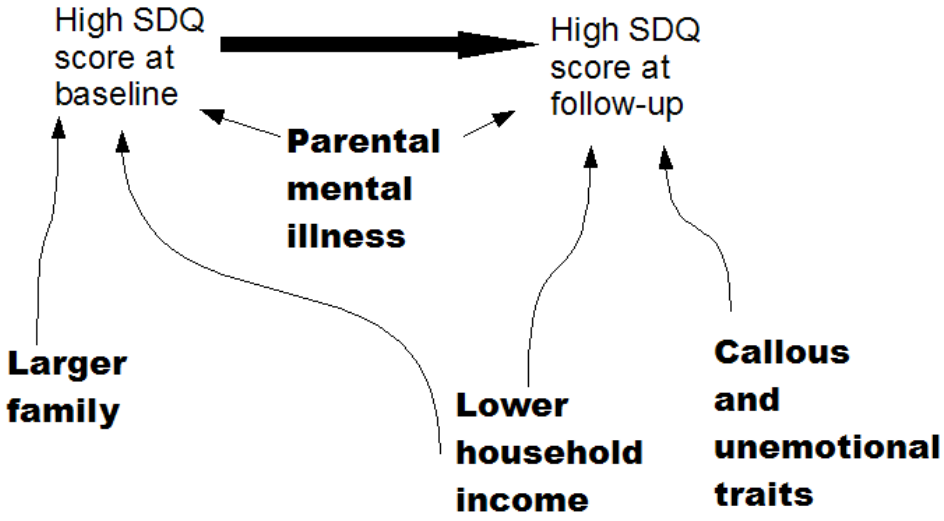


Figure 2.4 - Factors that predicted high SDQ scores.

Table 2.2 summarises the key strengths and weaknesses of this study.

STRENGTHS	WEAKNESSES
1. Large representative sample. 2. Two sources of information - parents and teachers - reduced bias. 3. Use of SDQ which is an established psychometric questionnaire.	1. The teachers' questionnaire not established as psychometric device (eg: validity and reliability). 2. Higher score of CUTs associated with groups over-represented in missing data - older children, larger families, poorer health, and Black and minority ethnicity. 3. Follow-up SDQ was posted whereas initial SDQ interview with parents was face-to-face.

Table 2.2 - Key strengths and weaknesses of this study.

EVALUATIVE FOOTNOTES

1. A community sample is a sample of the whole population as opposed to a clinical sample, who are individuals known to the authorities because they have sought treatment. A community sample overcomes the bias of a clinical sample (eg: misses those who have not sought treatment).

2. The population is divided into strata (based on, for example, social class, age, income, ethnicity) and within each strata a random sample is taken. Figure 2.5 shows a simple example of a random sample and a stratified random sample.

Population = 10 (under 35 = 70%; over 35 = 30%)
Sample = 4

A 31	E 52	I 75
B 27	F 27	J 61
C 25	G 12	
D 21	H 33	

Random sample = any 4 people; eg: E, J, I, A = 3 x over 35, 1 x under 35 (not representative)

Stratified random sample = any 3 x under 35, any 1 x over 35; thus 75% under 35 and 25% over 35 close to mirroring population

Figure 2.5 - Random sample and stratified random sample based on age.

3. (i) 10 496 approached

(ii) 7977 responded (76% of i)

(iii) 5770 teacher data (55% of i)

(iv) 4609 follow-up data (80% of iii, but 44% of i)

4. The SDQ measures conduct, emotional, hyperactive, and peer problems as well as a total score including pro-social behaviour.

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3. DIVERSE THEORIES OF SCHIZOPHRENIA

SCHIZOPHRENIA AS WAKING AND DREAMING TOGETHER

There are many theories about schizophrenia. Most focus on the biological/physical side, and a few on the mental/experiential side. Llewellyn (2002) has attempted to combine both the physical (brain) and the mental (mind) in suggesting that schizophrenia is "a global state of mind/brain 'trapped' between waking and dreaming: it's a disordered 'in-between' state, neither waking nor dreaming work properly because the mind/brain is attempting two, ultimately incompatible, sets of functions simultaneously" (p572).

Usually waking and dreaming are separate states because different neurochemicals are most active - eg: serotonin in waking and acetylcholine in dreaming (Llewellyn 2009).

Llewellyn (2009) outlined the particular way how schizophrenia is the "inbetween" state between the two (figure 3.1).

"The paradox is that waking and dreaming are our normal states of mind - we inhabit them everyday. But in 'schizophrenia' they become chaotically and chronically mixed up with devastating consequences" (Llewellyn 2009 p578).

COGNITIVE DEFICITS

Individuals with schizophrenia suffer from problems with attention, perception, memory, language, and decision-making (known as "cognitive deficits"). Generally it was assumed that these cognitive deficits were the consequence of schizophrenia. But recent research has suggested that cognitive deficits underlie the overt behaviour seen in schizophrenia (figure 3.2).

If this is so, cognitive deficits should be (Hoff and Kremen 2002) (table 3.1):

- Present before and at the onset of schizophrenia;
- Present in all sufferers;
- Present throughout even the schizophrenia is in remission;
- Present in non-schizophrenic biological relatives of sufferers.

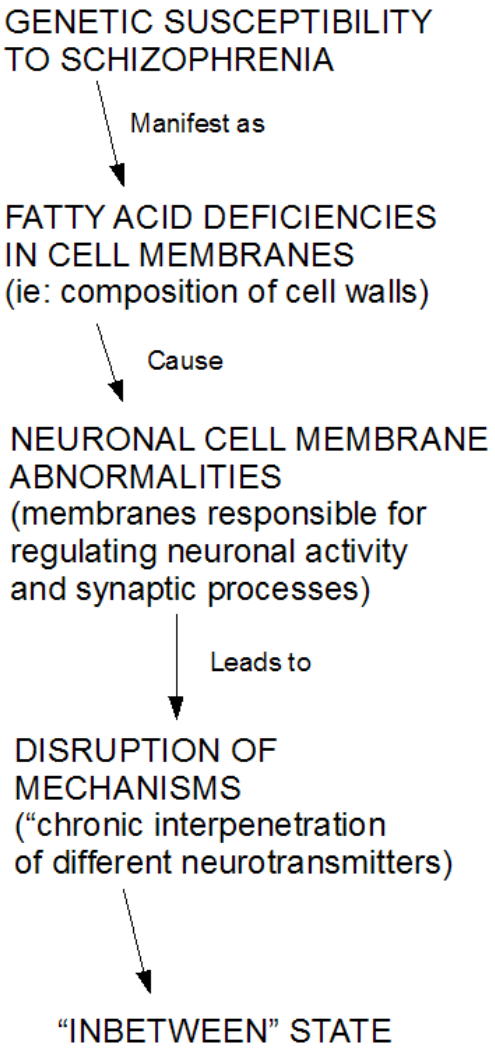

Figure 3.1 - Mechanism involved in schizophrenia as an "inbetween" state.

GENETIC BASIS
TO SCHIZOPHRENIA

↓ Produces

COGNITIVE DEFICITS

↓ Lead to

OVERT BEHAVIOUR AND
SYMPTOMS OF SCHIZOPHRENIA

Figure 3.2 - Cognitive deficits and schizophrenia.

PREDICTION	CONCLUSION FROM EVIDENCE
Cognitive deficits present at onset of schizophrenia	Executive function and memory deficits
Cognitive deficits present in all schizophrenia sufferers	Attention, memory and executive function deficits
Cognitive deficits present throughout the episode of schizophrenia	Similar cognitive deficits found in schizophrenics at first episode and long term sufferers, though cognitive deficits worsen with age
Cognitive deficits present in non-schizophrenic biological relatives of sufferers	Compared to controls: • Impaired performance in sustained attention task • Executive function impairment • Poorer verbal declarative memory test score (ie: verbal recall of items)

Table 3.1 - Conclusions about cognitive deficits underlying schizophrenia (Hoff and Kremen 2002) [1].

DOPAMINE AND SCHIZOPHRENIA

The link between too much of the neurotransmitter, dopamine and schizophrenia was established by a number of lines of evidence:

- Drugs (eg: anti-psychotics) which reduced dopamine also reduced the symptoms of schizophrenia (Potter and Hollister 1998);

- Drugs (eg: amphetamines) that increased dopamine produced behaviours similar to those in schizophrenia (Bell 1973);

- Schizophrenics produce more dopamine in response to amphetamines than non-schizophrenics (Abi-Dargham et al 1998);

- Post-mortems found excessive dopamine receptors in the brains of schizophrenics (Iversen 1979).

However, other research has challenged the excess dopamine theory. Also among other problems, dopamine only works in certain areas of the brain whereas the whole brain is involved in schizophrenia. Excess of dopamine is better at explaining positive symptoms. A revision of the theory suggested that reduced dopamine caused the

[1] Executive function refers to abilities to organise thoughts and behaviour. It is often tested by the Wisconsin Card Sorting Task (WCST).

negative and cognitive symptoms. But "improper dopamine balance cannot account for why one individual with schizophrenia responds almost completely to treatment, whereas someone else shows no apparent response" (Javitt and Coyle 2004).

McCarthy (2004) recently re-phrase the dopamine theory: "It is receptor sensitivity to dopamine (modulated by glutemate) and not the actual level of dopamine that determines the severity of a type-1 [2] schizophrenic episode" (p48).

Research in recent years has also looked at different types of dopamine receptors in the brain, like D2 and D4.

SCHIZOPHRENIA AS CAUSED BY AN INFECTION

The idea that mental illness was caused by an infection was first proposed in 1896 (Wenner 2008). Such an idea was subsequently rejected until the interest in infections like Toxoplasma gondii (Tg) as the cause of some schizophrenia.

Tg is, technically, a parasite mainly of cats, but it can live in other mammals including humans. In pregnant women, Tg can cross the placenta and lodge in brain tissue leading to Toxoplasma encephalitis with, for example, extreme head size (either too small or too large) and mental retardation (Torrey 2003).

Torrey (2003) reviewed the studies of the link between Tg and schizophrenia:

- 13 "early" studies published between 1953-1978 - 12 of the studies found that schizophrenics had higher levels of the Tg antibodies (a measure that Tg had been present in the body) than controls;
- No studies between 1979-1998;
- Six studies between 1999-2003 - All of them found more Tg antibodies in schizophrenics.

In a review of studies, particularly over a long period of time, there will be differences in the quality of studies (table 3.2).

[2] Positive symptoms (eg: hallucinations) as opposed to type-2 (negative symptoms; eg: catatonia).

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STUDIES BETWEEN 1953-1978	STUDIES BETWEEN 1999-2003
• Publication bias (or file-drawer problem) - negative studies probably not published. • Method of measuring antibodies less effective than newer studies. • Diagnostic criteria of schizophrenia varied or not specified. • Few details about healthy controls. • Most studies used in-patients, who may have been infected with Tg while hospitalised (eg: undercooked meat).	• Modern techniques for measuring Tg antibodies. • Modern diagnostic criteria for schizophrenia. • Control groups identified, and some matching. • Other physiological measures taken. • Mostly used first episode patients which ruled out Tg infection during hospitalisation.

Table 3.2 - Methodological strengths and weaknesses of old and new studies.

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4. DELIBERATE SELF HARMERS - WAITING FOR THEM TO COME VERSUS GOING OUT TO FIND THEM

Deliberate self harm (DSH) is a non-fatal act in which an individual deliberately injuries themselves. It tends to be a secretive behaviour, and so knowing how many people do it is not easy to find out. Two main techniques are used to answer this question:

- Self-referrals (eg: hospital attendees);
- General population surveys.

SELF-REFERRALS

Individuals who have harmed themselves, seek help and the number can be recorded. For example, Wright et al (1981) recorded the number of self-poisonings that presented at Dudley Road Hospital, Birmingham between January 1976 and December 1979. There were 2001 patients during this period. The drug ingested was recorded. Individuals were later questioned about their motives. The study found a large increase in cases of "West Indian" individuals over the period of the study. But it is difficult to know if this was a real increase or simply more individuals going to hospital (figure 4.1).

	REAL INCREASE		ARTIFICIAL INCREASE	
	Time A	Time B	Time A	Time B
	10 cases	15 cases	10 cases	10 cases
	↓	↓	↓	↓
Go to hospital	10	15	5	8

Figure 4.1 - Difference between real and artificial increase in cases.

Lockhart et al (1987) similarly recorded the self-poisonings at a west London district general hospital between November 1971 and February 1972, and September 1983 to June 1984. The incidence of self-poisonings halved between the two periods, while paracetamol use increased in self-poisonings. This fall was contrary to other studies of the time. However, the number of "West Indian" cases increased over the period. The two study periods are not really comparable because of the risk of random variations like the favoured hospital for admission. Again this makes it difficult to establish if

the changes are real or artificial (figure 4.2).

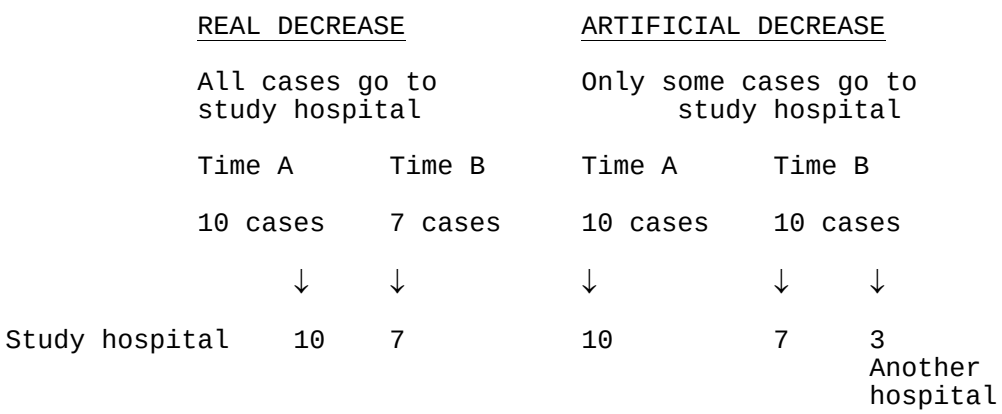

Figure 4.2 - Difference between real and artificial decreases based on hospital visited.

Table 4.1 gives the main strengths and weaknesses of using self-referrals to measure DSH.

STRENGTHS	WEAKNESSES
1. Actual cases. 2. Able to see the severity of self harm by the injury presented. 3. Accurate information can be recorded, like the drug involved in self-poisoning. 4. Study is cheaper as researchers wait for cases to come to them.	1. Misses individuals that do not seek help, or too mild to need medical assistance. 2. The decision of whether the injury was self harm may be made by another person like a doctor. Different doctors may record injuries differently. 3. How to distinguish between an accidental and a deliberate self-poisoning? Individuals may be embarrassed by seeking help and deny the real cause of the event. 4. Cannot distinguish real increases from artificial ones.

Table 4.1 - Strengths and weaknesses of using self-referrals to hospital to measure DSH.

GENERAL POPULATION SURVEYS

This method involves the use of questionnaires with the general population. It is possible to establish the prevalence of DSH in the population as a whole, and to compare the cases to the majority (non-cases) on social variables like gender and social class.

Hawton et al (2002) used forty-one schools in

Oxfordshire, Northamptonshire, and Birmingham in England for their survey. 6020 pupils aged fifteen and sixteen years completed the self-administered, anonymous questionnaire. Individuals were asked to describe any acts of DSH, and three independent raters categorised the behaviour using an agreed definition (table 4.2).

An act with a non-fatal outcome in which an individual deliberately did one or more of the following:

- Initiated behaviour (for example, self cutting, jumping from a height), which they intended to cause self harm;
- Ingested a substance in excess of the prescribed or generally recognised therapeutic dose;
- Ingested a recreational or illicit drug that was an act that the person regarded as self harm;
- Ingested a non-ingestible substance or object (Hawton et al 2002 p1208).

Table 4.2 - Definition of DSH used by Hawton et al (2002).

Lifetime prevalence of DSH was 13.2%, and in the previous year (focus of study), 6.9% met the agreed criteria for DSH. Importantly, only 50 of the 398 (12.6%) in the latter group attended hospital, and self-poisonings were more likely to go to hospital than cutting (which was the more common method used).

Analysis of social variables showed that DSH was more common in females, those females living with one parent, and with both sexes who had been bullied, and had been recently worried about their sexual orientation. Statistical analysis showed the factors associated with recent DSH:

- For female pupils - having friends who had recently self harmed, self harm by family members, drug use, depression, anxiety, impulsivity, and low self-esteem.

- For male pupils - having friends who had recently self harmed, self harm by family members, drug use, and low self-esteem.

There are certain weaknesses with this particular study:

1. The questionnaire was administered during school time, and so missed absent pupils. This might be important because other studies have found that DSH is more common among regular school truants (eg: Bjarnason and Thorlindsson 1994 in Iceland).

2. Respondents who did not provide a description of their DSH in the last year were not recorded. This produced a possible underestimation - 509 pupils reported DSH, but only 398 were defined by analysis of their description.

3. The lifetime prevalence as self reported in this study was double the figure from an English study (Meltzer et al 2001) using interviews with adolescents and their parents. How to know which study is more accurate (table 4.3)?

	HAWTON ET AL (2002)	MELTZER ET AL (2001)
Method	Self-administered questionnaires	Face-to-face interviews with adolescents and parents
More accurate	• Anonymous • Self-reported	• Information verified be parents • Interviewers can clarify terms
Less accurate	• Anonymity may encourage exaggeration as details cannot be verified • Misunderstanding of terms like DSH	• Embarrassment of face-to-face interviews and personal information • Parents may not know about or the true extent of DSH

Table 4.3 - Factors making two studies more or less accurate.

4. The study is a "one-shot" survey which measures the behaviour at a point of time. A longitudinal study with "multiple-shots" would be necessary to establish the pattern of causes for the behaviour.

Table 4.4 lists the overall strengths and weaknesses of using general population surveys to measure DSH.

STRENGTHS	WEAKNESSES
1. Able to gain a fuller picture of the prevalence of DSH in the general population. 2. Able to compare cases to non-cases on social variables. 3. Not dependent on individuals seeking help. 4. Able to use objective definition of DSH not dependent on subjective opinion of researcher.	1. Depends upon honesty of respondents. 2. Depends upon the accuracy of recall (eg: drug used in self-poisoning). 3. No independent verification of information. Some individuals may exaggerate the amount and severity of DSH as well as deny or understate it. 4. Expensive and time consuming study.

Table 4.4 - Strengths and weaknesses of using general population surveys to measure DSH.

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5. THE PHYSIOLOGY OF DEPRESSION: BRAIN AND BODY

BRAIN

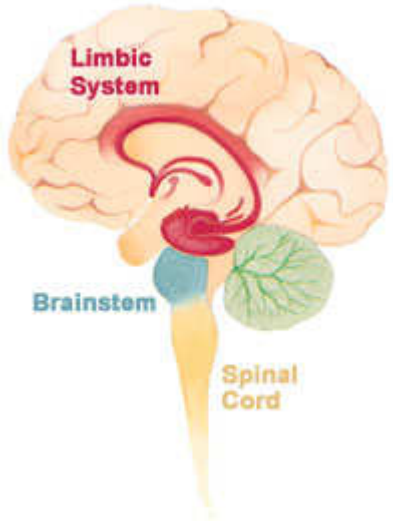
There are physiological changes during major depression in both the brain and the body.

Neuroimaging technology, developed in the last 15-20 years, allows researchers to "see" the brain of living individuals as they suffer from major depression. This has confirmed the already-established belief that depression has a physiological basis in the brain.

Depression is an emotional experience, and so the "emotion circuitry" of the brain is involved - ie: the limbic system (figure 5.1), and, in particular, the amygdala (figure 5.2) (table 5.1) (Gotlib and Hamilton 2008).

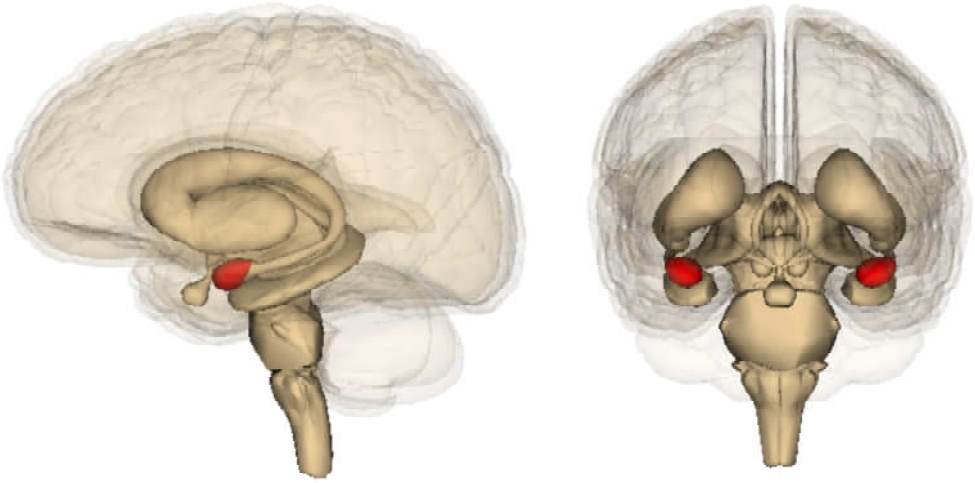
- No differences in amygdala volume in depressed and healthy individuals, but volume decreases with increasing episodes of depression.
- Elevated baseline amygdala activity and depression.
- Greater amygdala activity and depression.

Table 5.1 - Key differences in amygdala in depression.



(Source: US Federal Government; in public domain)

Figure 5.1 - Limbic system.



(Source: LifeScience Database; http://lifesciencedb.jp/?lng=en)

Figure 5.2 - Amygdala from two angles.

Another part of the limbic system involved is the anterior cingulate cortex (ACC), or, particularly, the ventral (bottom) or sub-genual ACC. Decreased resting state activity here in depressed individuals, and general reduced activity (Gotlib and Hamilton 2008).

Another area of the brain has also been examined - the dorsolateral prefrontal cortex (DLPFC). This area of the brain is involved in cognitive control of behaviour. The high level of rumination in depression is seen as a problem in cognitive control, and is because of less activity in the DLPFC (Gotlib and Hamilton 2008).

Overall, "there are functional abnormalities of neural structures implicated in the experience, expression and regulation of emotion in depression" (Gotlib and Hamilton 2008 p160). But what is the relationship between these different areas of the brain in depression? This is a question that is taxing researchers. For example, the greater activity in the amygdala reduces the activity in the DLPFC, which now cannot control the amygdala, and a vicious circle is created (Mayberg et al 1999 quoted in Gotlib and Hamilton 2008).

BODY

From a different viewpoint, Chen et al (2009) observed the higher prevalence of depression in

individuals with autoimmune diseases, and the presence of suppressed immunity in depressed individuals [3]. They argued that rather than one causing the other in each case, these are evidence that depression is an autoimmune disease caused by various autoantibodies (figure 5.3). In autoimmune diseases, the immune system attacks the body tissue mistakenly as it would invaders (pathogens).

Autoantibodies (antibodies attacking the body) may cause depression through different physical mechanisms:

- Attacks on the neuroendocrine system [4] (eg: hypothalamic-pituitary-adrenal axis; figure 5.4) causing dysfunction;
- Disturbing neurochemical processes, like neuropeptides [5];
- Binding to certain areas of the brain, like cingulate cortex, and affecting its working.

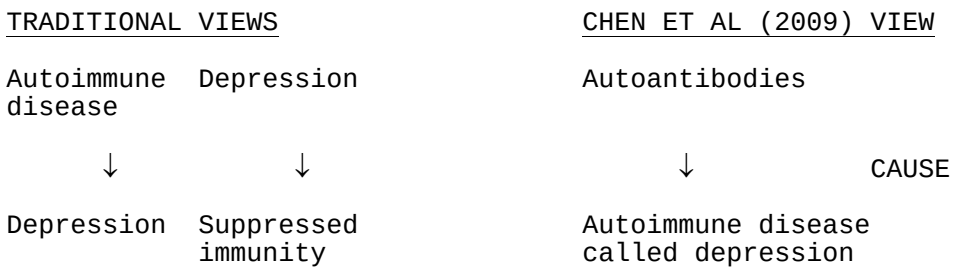


Figure 5.3 - Relationships between depression and the immune system.

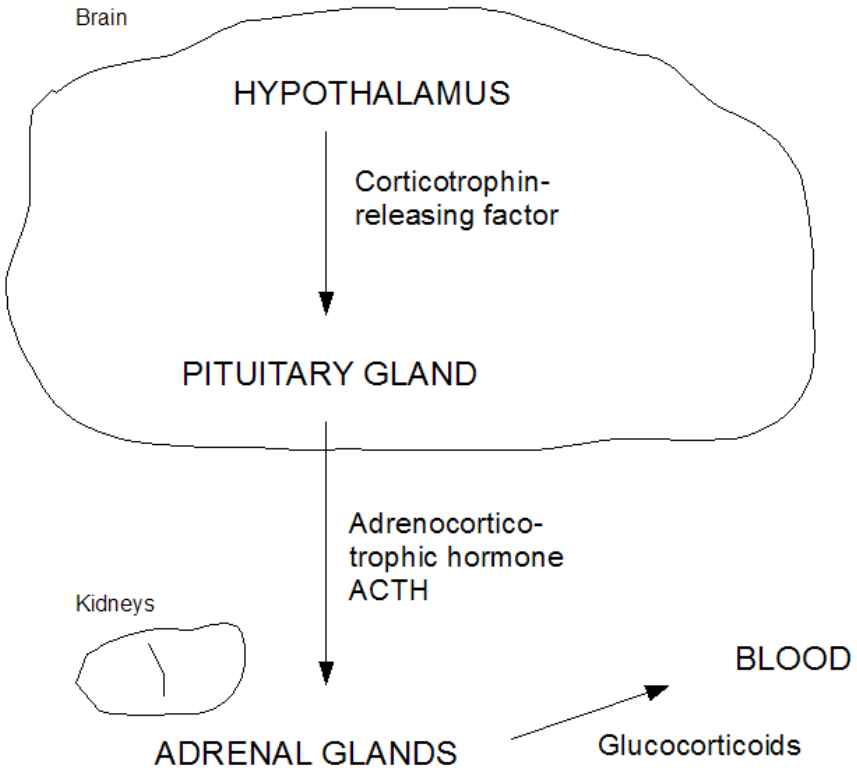
CONCLUSIONS

Are these physiological changes in depression described above observations of the same thing from different angles, or completely different aspects? There is a lot of interest in the brain, while the autoimmune disease theory is new.

[3] However, there is no consensus of opinion on the impact of depression upon immune functions with inconsistent findings (Evans et al 2000).

[4] Neuroendocrine system is term for interaction of the nervous system and the endocrine (hormone) system of the body.

[5] General details about neuropeptides at http://www.neuropeptides.nl/.



(Adapted from Song and Leonard 2000)

Figure 5.4 - Hypothalamic-pituitary-adrenal (HPA) axis.

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6. SUMMER BIRTHS AND SCHIZOPHRENIA

Astrology places importance on the individual's birth date for determining their lives (through the workings of planets). Science has traditionally rejected such ideas.

However, of recent, "season of birth effects" have been reported for academic achievement (eg: Fieder et al 2006), personality traits (eg: Chotai et al 2001 [6]), and schizophrenia. But physiological mechanisms are being proposed for these effects - eg; exposure to influenza in the womb and its effect on the foetus.

Torrey et al (1997) reported that individuals with schizophrenia were more likely to have been born in the winter months ("winter birth excess"). A 5-8% winter-spring excess for schizophrenia, mania, and bipolar disorder, and a December-March excess for schizoaffective disorder.

Kirkpatrick et al (2002) subsequently noted a June/July (and summer) birth excess in the non-tropical northern hemisphere for individuals with negative symptoms in schizophrenia (eg: apathy, decreased sociality). This "deficit schizophrenia" is shown in greater anhedonia (lack of enjoyment from pleasurable experiences) but less depression than usual type II schizophrenia [7] (Kirkpatrick et al 2008).

Kirkpatrick et al (2002) analysed the month of birth of all patients diagnosed with schizophrenia, schizoaffective disorder, delusional disorder, mania, drug-induced psychotic disorder, or acute, transient or unspecified psychotic disorder (ICD-9 criteria) in Dumfries and Galloway, Scotland (figure 6.1) between 1979 and 1998. Of these deficit schizophrenia was assessed by the Schedule for the Deficit Syndrome (Kirkpatrick et al 1989).

This produced sixty-five deficit and 277 non-deficit schizophrenia patients. Individuals with deficit schizophrenia were found to be born in the summer months more often than non-deficit schizophrenics, and the general population of the area (table 6.1).

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[6] Those born between February and April (Spring born) in Sweden were significantly more likely to show high novelty seeking behaviour in females and high persistence in males than those born between October and January (Winter born). This finding has not been replicated (Kirkpatrick et al 2008).

[7] Type I schizophrenia refers to "positive" symptoms like hallucinations (similar to mania), and type II to "negative" symptoms like inactivity and flatness of emotion (similar to depression) (Brewer 2001).

MONTHS	DEFICIT SCHIZOPHRENIA BIRTHS (%) (N = 65)	ALL BIRTHS IN DUMFRIES AND GALLOWAY (%) (n = 1809)	ODDS RATIO DEFICIT TO NON-DEFICIT SCHIZOPHRENICS
June-August	37	26 ($p<0.05$)	2.01 ($p<0.03$)
June-July	28	17 ($p<0.02$)	2.53 ($p<0.01$)
July-August	26	19 (ns)	2.13 ($p<0.04$)

(ns = not significant at p = 0.05 level)

(After Kirkpatrick et al 2002)

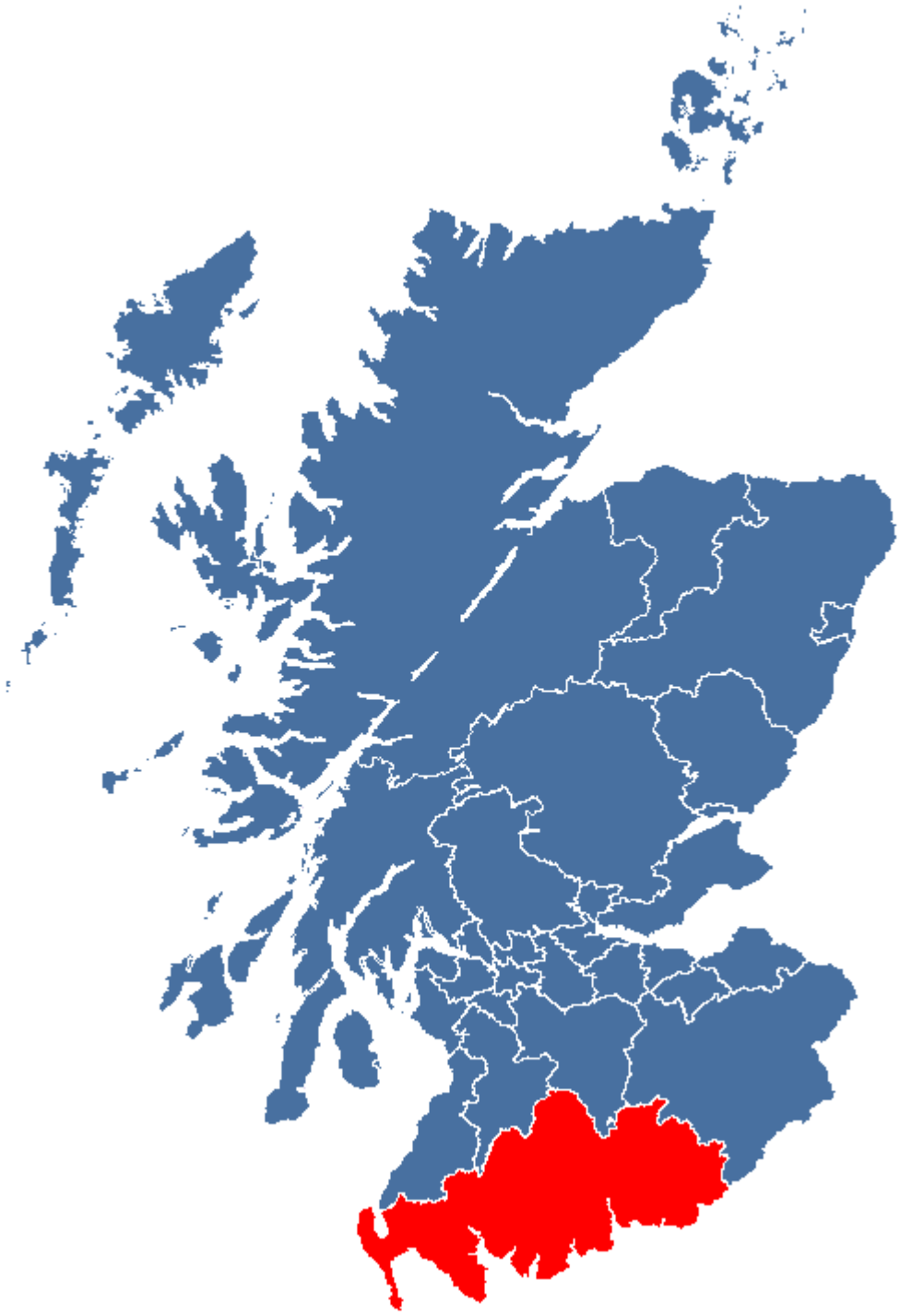
Table 6.1 - Amount of births in summer months.

Birth in the summer would mean pregnancy during the winter, particularly when viral infections are more common and have the greatest effect during the second trimester (3-6 months gestation age).

Kirkpatrick et al (2008) took the research a step further as they were interested to see if characteristics of deficit schizophrenia were evident (to a lesser degree) in the general population born in June and July. 426 US undergraduates were recruited, and given three of the Chapman psychosis proneness scales (Eckblad and Chapman 1983) and the Beck Depression Inventory (BDI) (box 6.1). The subtraction of he BDI score from the Social Anhedonia scale produced the "Proxy for the Deficit Syndrome" (PDS) score.

Participants born in June-July (n = 25) had significantly higher (p = 0.0037) PDS scores than any other months, and so did males than females (p = 0.002).

Table 6.2 lists the key weaknesses of this study.



(Source: Interfector)

Figure 6.1 - Outline map of Scotland showing position of Dumfries and Galloway.

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1. Chapman psychosis proneness scales: true or false self-reported statements about the self.

i) Magical Ideation - measures belief in unusual associations in the world with 30 items; eg: "At times I have felt that a professor's lecture was meant especially for me" (box 6.2);

ii) Perceptual Aberration - measures experience of perceptual distortions; eg: "I have sometimes felt confused as to whether my body was really my own";

iii) Social Anhedonia - measures pleasure experienced from social interactions; eg: "I prefer hobbies and leisure activities that do not involve other people".
The scores were combined in the PER-MAG scale.

2. Beck Depression Inventory (Beck et al 1961) - self-reported measure of depression containing 21 items; eg: Mood - "I do not feel sad" (0), "I feel blue or sad" (1), "I am blue or sad all the time and I can't snap out of it" (2a), "I am so sad or unhappy that it is very painful" (2b), "I am so sad or unhappy that I can't stand it".

Box 6.1 - Measures used by Kirkpatrick et al (2008).

1. Self-reported measures used, but they were standardised.

2. No control for place of birth, and so birth season is different in the northern and southern hemispheres.

3. Only a small number born in June-July.

4. Convenience sample used (ie: students at university), which is not representative of the general population.

Table 6.2 - Key weaknesses of study by Kirkpatrick et al (2008).

1. Some people can make me aware of them just by thinking about me.
2. I have had the momentary feeling that I might not be human.
3. I have sometimes been fearful of stepping on side- walk cracks.
4. I think I could learn to read other's minds if I wanted to.
5. Horoscopes are right too often for it to be a co- incidence.

6. Things sometimes seem to be in different places when I get home, even though no one has been there.
7. Numbers like 13 and 7 have no special powers.
8. I have occasionally had the silly feeling that a TV or radio broadcaster knew I was listening to him.
9. I have worried that people on other planets may be influencing what happens on earth.
10. The government refuses to tell us the truth about flying saucers.

11. I have felt that there were messages for me in the way things were arranged, like in a store window.
12.1 have never doubted that my dreams are the products of my own mind.
13. Good luck charms don't work.
14. I have noticed sounds on my records that are not there at other times.
15. The hand motions that strangers make seem to influence me at times.

16. I almost never dream about things before they happen.
17. I have had the momentary feeling that someone's place has been taken by a look-alike.
18. It is not possible to harm others merely by thinking bad thoughts about them.
19. I have sometimes sensed an evil presence around me, although I could not see it.
20. I sometimes have a feeling of gaining or losing energy when certain people look at me or touch me.

21.1 have sometimes had the passing thought that strangers are in love with me.
22. I have never had the feeling that certain thoughts of mine really belonged to someone else.
23. When introduced to strangers, I rarely wonder whether I have known them before.
24. If reincarnation were true, it would explain some unusual experiences I have had.
25. People often behave so strangely that one wonders if they are part of an experiment.

26. At times I perform certain little rituals to ward off negative influences.
27. I have felt that I might cause something to happen just by thinking too much about it.
28. I have wondered whether the spirits of the dead can influence the living.
29. At times I have felt that a professor's lecture was meant especially for me.
30. I have sometimes felt that strangers were reading my mind .

(Eckblad and Chapman 1983 pp216-217)

Box 6.2 - Magical Ideation Scale.

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7. STIMULANT MEDICATION FOR ADHD IN CHILDREN

In the USA it is estimated that 9% of boys and 4% of girls aged 6-17 years are taking stimulant medication (psycho-stimulants), like methylphenidate ("Ritalin", "Concerta") or amphetamines (eg: "Adderell") to combat Attention Deficit Hyperactivity Disorder (ADHD) (Higgins 2009). Between 1990 and 1995 prescription of stimulants for ADHD increased dramatically in North America (2.5-fold in the USA and 5-fold in Canada) (Phillips 2006).

The discovery of the effectiveness of stimulants on hyperactive children occurred by accident in the 1930s (Bradley 1937). Charles Bradley at a private children's home in Rhode Island gave a new amphetamine, Benzedrine, to children with behavioural difficulties. But for a long time only children hospitalised for severe mental disorders were given them (Mayer and Rafalovich 2007).

Methylphenidate was first synthesised by Swiss pharmaceutical company, J.R.Geigy, after the 2nd World War, and given the brand name, "Ritalin", it was approved for use in the USA in 1961 (Mayer and Rafalovich 2007).

Singh (2008) outlined four positions in the "public debate over the validity of he diagnosis, the root causes of ADHD and the ethics of treating children with psychotropic drugs":

- ADHD has a biological cause which only medication really can deal with;
- ADHD is a combination of biological and social causes, and medication is thus not automatically the best way to help sufferers;
- ADHD has environmental causes (eg: food additives) and prevention is better than medication;
- All the above positions accept the validity of ADHD, whereas this position is sceptical that it is a "real disorder", and thus sees medication as completely unacceptable.

ARGUMENTS FOR USE

1. In ADHD areas of the frontal cortex are responsible for attention and self-control are less active, and the stimulants increase the activity. In terms of self-control, it is like a brake, and less activity in the brain means less pressure on the brake, while increased

brain activity equals increased pressure on the brake (figure 7.1). Thus impulsiveness is reduced.

Stimulants allow greater dopamine activity in the brain by deactivating the processes that suck up excess dopamine after a synapse (biochemical process in the brain).

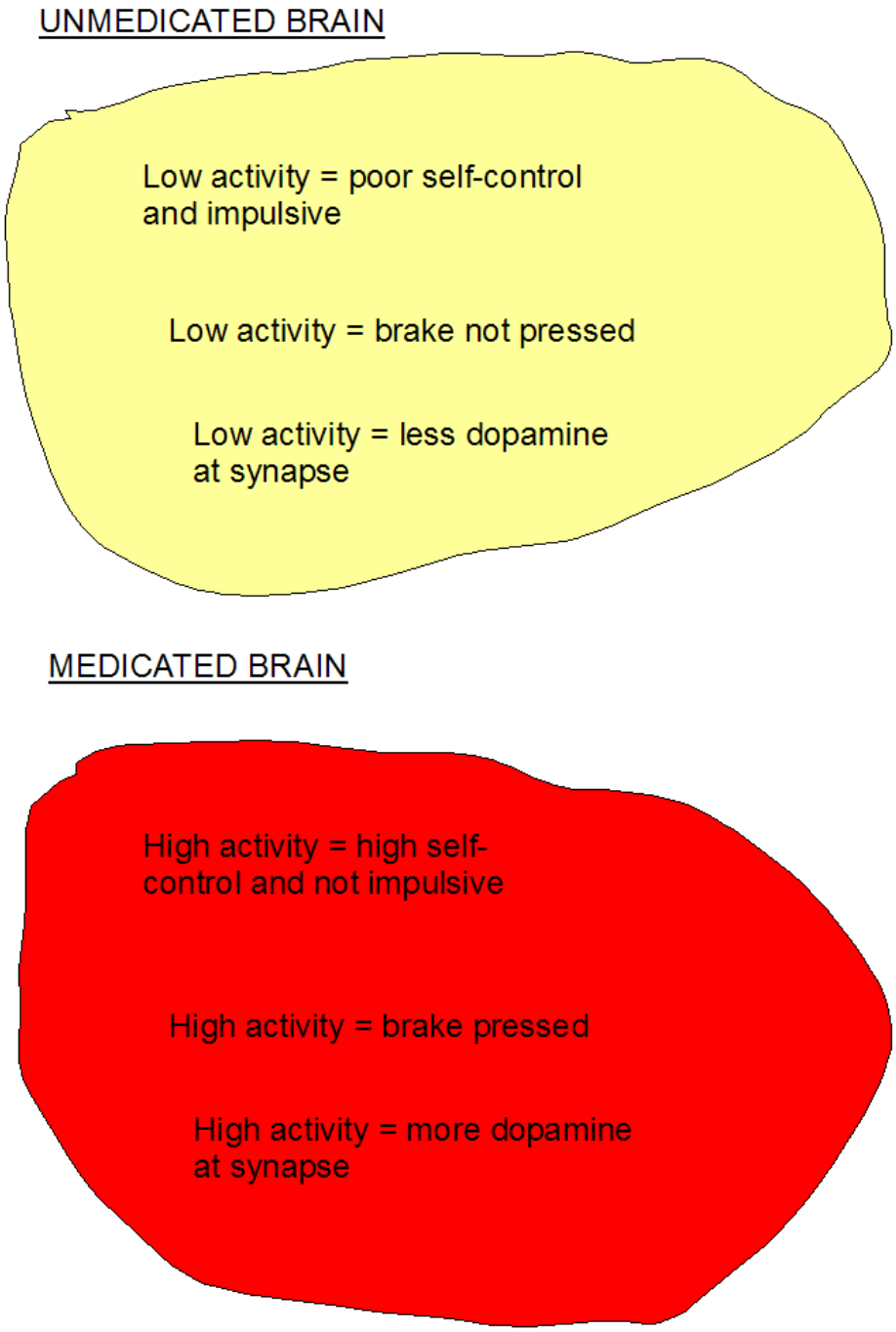

Figure 7.1 - Brake on self-control and brain activity.

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2. Large-scale studies have shown the efficacy/effectiveness [8] of medication. The NIMH Collaborative Multi-Site Multi-Treatment Study of Children with ADHD (MTA study) compared 579 ADHD children aged 7-10 years in the USA on medication, intensive behavioural treatment, and a combination of both of these. Nineteen measures of behaviour were used, and children showed improvements on medication and combined treatments over fourteen months of the study (Rubin and Smith 2001).

Generally, studies show a 70-75% probability of improvement for some children (Chu 2003b).

Faraone et al's (2006) meta-analysis found twenty-nine clinical trials since 1979 comparing medication to placebo for ADHD. Overall, all medications (fifteen different drugs) were significantly better than placebo, but immediate-release stimulants and long-acting stimulants were significantly more effective than non-stimulants like tricyclic anti-depressants. But studies used different outcomes measures of behaviour (17 different found).

3. ADHD is an accepted diagnostic category and mental disorders for psychiatrists with core symptoms of inattention, hyperactivity, and impulsiveness. The key aspect of ADHD is hyperactivity, and individual differences in this behaviour have an underlying physical basis (Taylor 2004).

There are clear genetic, neuroanatomical (eg: reduced blood flow in frontal cortex) and neurochemical bases (Chu 2003a).

4. ADHD is a risk factor for adult problems including involvement in crime. Medication as a child (and as an adolescent) would reduce these risks.

5. Stimulants have been prescribed for many years, since the 1950s in the USA, without major consequences. Many neuroimaging studies have not found differences between the brains of children and adolescents taking stimulants and those not (Higgins 2009).

6. Even children without ADHD given stimulants become more attentive, and gain higher test scores (Gazzaniga

[8] "Efficacy" refers to outcomes under ideal conditions, while "effectiveness" is how well it works under practice conditions (Zito and Provenzano 1995).

2005). But Singh (2008) admitted: "How stimulants improve focus, attention and impulsive behaviour is still poorly understood" (p960).

However, the common use of stimulants with children is not the same as popular use by university students and academics as "performance enhancers" (Singh 2008).

ARGUMENTS AGAINST USE

1. Concern about long-term effects of the drugs upon the developing brain and body. For example, a large US study has found that medicated 7-10 year-olds grew at a slower rate than unmedicated children (Higgins 2009). While studies with rhesus monkeys injected with amphetamines for twelve weeks showed working memory deficits that lasted for three years after exposure (Higgins 2009).

But the animal studies are criticised for using larger doses than children would receive, and for injecting the drug which children do not do either. Ricaurte et al (2005) overcame these criticisms by giving baboons and squirrel monkeys appropriate doses orally of amphetamines for four weeks. Subtle differences in the levels of dopamine, particularly in the striatum area, were found compared to controls.

Furthermore, children are often given "drug cocktails" (ie: more than one type of drug) and despite the lack of safety data about such practices (Singh 2008).

Meanwhile, "Children are not small adults, nevertheless, most of the psychotropic drugs that are prescribed to children have only been tested on adults" (Singh 2008 p962).

2. Concern about side effects of use like the development of other mental disorders. In adulthood "Having ADHD is itself a risk factor for other mental health problems, but the possibility also exists that stimulant treatment during childhood might contribute to the high rates of accompanying diagnoses" (Higgins 2009 p40).

Animal studies show mood alteration after long-term use of methylphenidate in infancy. For example, juvenile rats injected with small amounts of the drug, as adults were less responsive to rewards and more sensitive to stressors than control rats (Higgins 2009).

The usual side effects are seen as mild, like appetite loss and insomnia. Though since February 2007, US authorities have placed warnings on the packets about, for example, cardiovascular problems (Singh 2008).

3. The risk of addiction. A report in the USA in 2006 suggested that three-quarters of a million teenagers and young adults showed signs of addiction to methylphenidate (Higgins 2009).

In terms of chemical structure, methylphenidate is similar to cocaine. Kim et al (2009) injected mice daily for two weeks with one of these two substances. Similar changes were found in the brain, particularly in the nucleus accumbens. Also changes in certain proteins heighten the sensitivity to cocaine.

Berbatis et al (2002) argued, with particular reference to Australia, that there is a significant correlation between "licit" prescriptions of amphetamines for ADHD and illicit amphetamine use.

4. Non-medication treatments for ADHD, like Behaviour Modification, and mental exercises, can be as effective as medication in the long-term (and without the side effects of medication) (Kraft 2006).

5. Over-use and inappropriate use of medication. ADHD is increasingly diagnosed for moderate to mild inattention, which is within the normal range of attention for children. So medication is being subsequently prescribed inappropriately.

Part of the reason for this comes from the manufacturers of these drugs. Phillips (2006) felt that in the USA, in particular, there is "the organised penetration of the pharmaceutical industry associated with ADHD into the education domain". This involves the marketing of drugs like Ritalin in the same way as any other product, especially aimed at teachers and parents.

6. Concerns about the medicalisation of society and the ethics of prescribing powerful stimulants to large numbers of children.

The concept of ADHD is challenged as a cultural construct: "There is no established prognosis, and association and cause frequently are confused in the literature" (Timimi 2004 p8).

An extreme anti-ADHD position is taken by Baughman (2003), who saw ADHD as "a total fraud leading to the medical victimisation of millions of previously normal, if troubled, mis-educated, children across the United States" (p93).

"By conceptualising problems as medically caused we end up offering interventions ..that teach ADHD-type behaviour to the child. ADHD causes 'tunnel vision' in the system, making it more difficult to think about

context, leading to interpersonal issues being marginalised. ADHD scripts a potentially life-long story of disability and deficit, resulting in an attitude of a 'pill for life's problems'" (Timimi 2004 p8).

While studies about medication tend to ignore the children's views and age-related "treatment-related decision-making capacity" (Singh 2008).

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8. OBSTETRIC COMPLICATIONS AND SCHIZOPHRENIA

The search for the cause of schizophrenia has included the time in the womb and immediately after birth. Risk factors could include pre-eclampsia (pregnancy-induced hypertension), birth complications, and low birth weight. Most studies are retrospective using individuals diagnosed with schizophrenia as adults and looking backwards. So, for example, maternal recall of the pregnancy and birth can be the way used to establish complications. This way risks bias and inaccuracy.

Dalman et al (1999) made use of the extensive records of the National Birth Register in Sweden for all children born between 1973 and 1977. Midwives, for example, collect information during pregnancy, and about the birth and the newborn.

The researchers were interested in three aspects:

i) Foetal malnutrition - problems in the supply of oxygen or nutrients to the foetus as shown by small physical size for gestational age;

ii) Extreme pre-maturity - delivery before week 33 of gestation;

iii) Complications at delivery - eg: lack of oxygen, or bleeding.

Of the 507 516 children born in the study period, 238 were diagnosed with schizophrenia between 1987 and 1995.

It was found that obstetric complications as recorded by medical staff at the time were associated with development of early onset schizophrenia, in particular, pre-eclampsia, premature birth, emergency caesarean section, low birth weight (less than 1500g). All of these complications can produce brain damage. Older mothers (40 years old or more) were only a risk for males.

Individually these factors increased the risk of schizophrenia two to three-fold, whereas the risk was nearly eight times greater when the mother had a psychotic illness at some stage during her adult life.

Though this was a large-scale study, the number of individual cases were small; eg; eleven cases with pre-eclampsia developed schizophrenia, and two with very low birth weight.

From a slightly different angle, Myhrman et al

(1996) found that schizophrenia was twice as common in Finland in unwanted pregnancies. Obstetric complications provide a possible physiological link for schizophrenia in the form of brain damage, but why unwanted pregnancies? There could be a physiological link in the womb in terms of the mother's failure to at sufficiently or the stress of the pregnancy with the altered hormonal regulation, for example.

Herman et al (2006) used data from the Prenatal Determinants of Schizophrenia (PDS) Study [9] which involved live births between 1959 and 1967 in part of California, USA. It was a prospective study.

"Unwanted pregnancy" was measured by the question, "how do you feel about having a baby now?" at first contact with the pre-natal clinic. The open-ended responses were coded as "strongly favourable", "moderately favourable", "ambivalent", "negative", and "other" (no attitude expressed). Schizophrenia spectrum disorder (SSD) [10] was measured by DSM-IV diagnostic criteria.

There was a significant association between ambivalent and negative attitudes towards the pregnancy by the mother and diagnosis of the offspring with SSD. But this significant result is not necessarily an accurate picture of reality. 1499 women (out of 7795) rated their attitude towards the pregnancy as ambivalent or negative, and only sixteen of their offspring were diagnosed with SSD. So the vast majority of unwanted pregnancies did not lead to SSD. "Rather, it is likely to interact with other vulnerability factors to increase schizophrenia risk in certain susceptible individuals" (Herman et al 2006 p609).

Table 8.1 summarises the strength and weaknesses of this study.

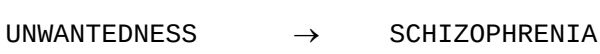
[9] Details of design in Susser et al (2000).

[10] This covers schizophrenia, schizoaffective disorder, delusional disorder, psychotic disorder not otherwise specified, and schizotypal personality disorder.

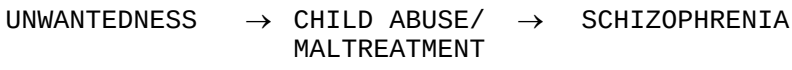
STRENGTHS	WEAKNESSES
1. Prospective study that does not depend on mother recalling her attitude towards the pregnancy. 2. Attitude towards pregnancy measured by open-ended question, and researchers coded the responses. 3. Large cohort of births (7795 participants out of 12 094 total births in research period). This was all women who received pre-natal care from Kaiser Foundation Health Plan. 4. Individuals diagnosed with SSD using ICD-9 criteria were rediagnosed by the researchers using DSM-IV criteria. 5. Detailed data about demographics collected at time of pregnancy. The women were representative of the California area population of the time, in terms of race, education and occupation. 6. The median follow-up was 30 years.	1. It was a correlational study that compared the relationship between two variables at two points in time. It could not establish causation (figure 8.1). 2. The number of SSD cases was small - 70. 3. "Unwanted pregnancy" was measured by a single question at one point in time when the women first visited the clinic. Many of the women may have come to terms with it and not held a negative attitude throughout the pregnancy. 4. No data collected on biological parent(s)' history of mental illness. 5. No data on the family environment after birth and during childhood, which could have been the cause of the schizophrenia. 6. Limited understanding generally about the link between unwantedness and schizophrenia.

Table 8.1 - Strengths and weaknesses of Herman et al (2006) study.

1. Unwantedness causes schizophrenia (cannot say because it is correlation)



2. Unwantedness causes another factor (unseen or unrecorded) that causes schizophrenia (study could not tell because did not study inbetween two points of research)



3. Another factor (unseen or unrecorded) independent of unwantedness causes schizophrenia (study could not tell because did not measure family history of mental illness, for example)

Figure 8.1 - Three relationships between unwantedness and schizophrenia that appear as correlations.

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9. ATTITUDES TO CHOCOLATE QUESTIONNAIRE AND CRAVING

The term "addiction" is used widely today, and not only in relation to drugs, but also behaviours like exercise or gambling. Addiction involves a problematic relationship to the addictive substance including an inability to resist intake, a craving for it, and often excessive intake. It is possible to include chocolate in the list of substances of addiction.

Benton et al (1998) developed a questionnaire to measure attitudes towards chocolate consumption and craving for it [11]. They initially asked 226 female and 104 male undergraduates in their teaching groups at the University of Wales in Swansea [12] to respond to eighty statements about chocolate [13]. The statements covered areas like difficulty in controlling chocolate consumption, expected relief of negative mood from eating it, and experience of craving.

For each statement, the respondents were offered a line 100mm long with "very much like me" at one end and "not at all like me" at the other end. The respondents placed a mark on the line that reflected their answer [14]. Each millimetre was converted into a point later.

The responses were subject to factor analysis (using principal component analysis and varimax rotation) [15]. This produced three underlying factors:

i) "Craving" - preoccupied with chocolate and a compulsion to eat it. Statements here include, "My desire for chocolate often seems overpowering" and "Chocolate often preys on my mind".

ii) "Guilt" - negative experiences linked to eating chocolate; eg: "I feel unattractive when I have eaten chocolate" or "I feel guilty after eating chocolate".

---

[11] In a Internet-based questionnaire of approximately 3000 people, Parker and Crawford (2007) found a link between chocolate craving and "atypical depression" symptoms.

[12] This was an opportunity sample based on who is available in the psychology classes. Being undergraduates the mean age was 21.6 years old. Thus the sample was not representative of the general population. However, this method of sampling is convenient.

[13] All students in the groups filled in the questionnaire to avoid self-selection on the basis of interest in chocolate. Does this mean that the respondents did not have the right to withdraw or refuse?

[14] This is a variation of the Semantic Differential Scale (Osgood et al 1957).
Advantages - avoids use of language and associated influence of words on answers, and measures feeling part of attitudes.
Disadvantages - tendency towards middle point, and difficult to assess without the anchor of words.

[15] Factor analysis aims to find the underlying factors or constructs that explain the relationship between scores on different items of the questionnaire. There are a number of different techniques of factor analysis.

iii) "Functional approach" - this is a pragmatic attitude towards chocolate; eg: "I eat chocolate only when I am hungry" or "I eat more chocolate in the winter when it is colder".

The twenty-four statements that were most discriminating were included in the "Attitudes to Chocolate Questionnaire" (ACQ) (table 9.1).

1. I eat chocolate to cheer me up when I am down. (C)
2. My desire for chocolate often seems overpowering. (C)
3. I feel unattractive after I have eaten chocolate. (G)
4. I often feel sick after I have eaten chocolate. (G)

5. I eat chocolate as a reward when everything is going really well for me. (F)
6. I am often on one kind of diet or another. (G)
7. The thought of chocolate often distracts me from what 1 am doing (eg: watching TV). (C)
8. I usually find myself wanting chocolate during the afternoon. (C)

9. I consider chocolate to be high in fat and to be of poor nutritional value. (G)
10. After eating chocolate 1 often wish I hadn‘t. (G)
11. I feel guilty after eating chocolate. (G)
12. I eat chocolate only when 1 am hungry. (F)

13. Chocolate often preys on my mind. (C)
14. I feel unhealthy after I have eaten chocolate. (G)
15. I always look at the calorific value of a chocolate snack before 1 eat it. (G)
16. If I resist the temptation to eat chocolate I feel more in control of my life. (G)

17. Nothing else but chocolate will satisfy my chocolate cravings. (C)
18. Even when I do not really want any more chocolate I will often carry on eating it. (C)
19. I eat chocolate to keep my energy levels up when I am doing physical exercise. (F)
20. I eat more chocolate in the winter when It 1s colder. (F)

21. I often go into a shop for something else and end up buying chocolate. (C)
22. I feel depressed and dissatisfied with life after eating chocolate. (G)
23. I often eat chocolate when I am bored. (C)
24. I like to indulge in chocolate. (C)

(C = craving, G = guilt, F = functional)

(After Benton et al 1998 table 2 p516)

Table 9.1 - Attitudes to Chocolate Questionnaire.

Further analysis of the responses showed gender differences - female students scored significantly higher on Craving items and Guilt items than males (figure 9.1).

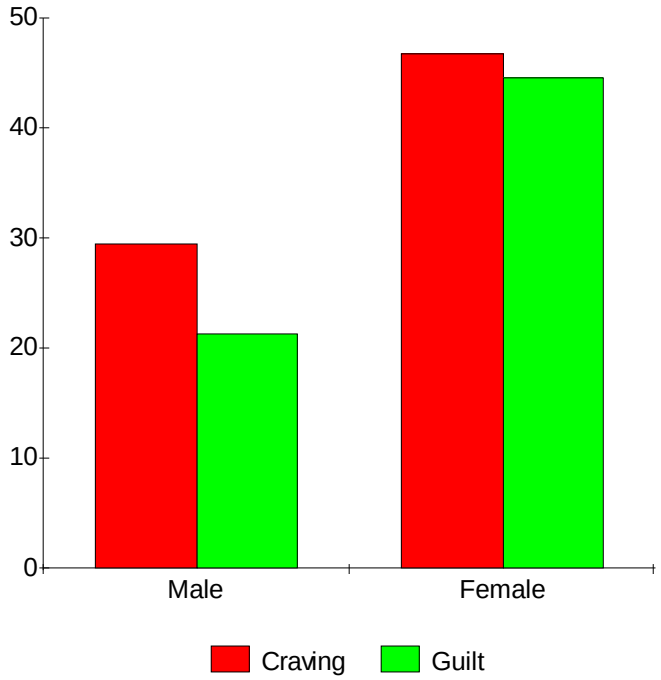

Figure 9.1 - Mean scores for male and female students on Craving and Guilt items.

Four groups of female respondents were created for comparison - high Craving/high Guilt (HC/HG), high Craving/low Guilt (HC/LG), low Craving/high Guilt (LC/HG), and low Craving/low Guilt (LC/LG) [16]. The number of chocolate bars consumed per week was most in the HC/LG group (mean of 8) followed by HC/HG (mean of 6). The mean for the other two groups was approximately two chocolate bars per week.

Response to the statement, "I sometimes force myself to be sick after eating chocolate" varied between the groups. It was strongest in the HC/HG group followed by LC/HG. Agreement with "Even when I do not really want chocolate I carry on eating it" and "I sometimes do not eat chocolate for several days and then eat a large amount in one go" showed significant differences between the groups (table 9.2).

A sub-group of forty female and twenty-seven male students were set a task of pressing a space bar on a computer for a certain number of times to gain rewards of chocolate buttons. The reward (reinforcement) was given initially for two presses, then four, eight, sixteen and so on. This is a progressive-ratio schedule. When the

[16] Scores in the top and bottom 30% of the range were used for these groups, but the groups were not equal sized - HC/HG (n = 34), HC/LG (14), LC/HG (7), and LC/LG (19).

STATEMENT	HC/HG	HC/LG	LC/HG	LC/LG
"I sometimes force myself to be sick after eating chocolate"	15	2	10	2
"Even when I do not really want chocolate I carry on eating it"	90	60	20	20
"I sometimes do not eat chocolate for several days and then eat a large amount in one go"	75	30	40	20

Table 9.2 - Approximate mean scores for four groups on three statements.

participants stopped (ie: choosing to finish the task) was the measure of their effort to gain chocolate. Individuals with high Craving scores were more likely to continue longer on this task. There was no significant relationship with the other two factors (Guilt and Functional).

Overall, a high Craving score predicted greater consumption of chocolate and greater willingness to work for it, while a high Guilt score was associated with the behaviours in table above (binge and vomit behaviours). Craving is a key aspect of addiction to drugs of abuse.

Cramer and Hartleib (2001) gave the ACQ to 173 male and 537 female first-year undergraduate psychology students at the University of Windsor, Canada as part of a course requirement. The response choices were changed to a five-point Likert scale [17] (1 = "strongly disagree" to 5 = "strongly agree") for convenience.

Confirmatory factor analysis of the scores only found two underlying factors - Craving and Guilt [18].

Female students had significantly higher scores on Craving items and Guilt items than male students (figure 9.2).

A sub-group of students completed the ACQ again three months later. The comparison of their scores for test-retest reliability [19] produced correlations of $r = 0.69$ (Craving items) and $r = 0.68$ (Guilt items) (both $p<0.001$).

High Craving scores were found to correlate with other measures of depression and obsession, and with low

[17] The Likert scale (Likert 1932) offers a choice of responses in simple language.
Advantages - simple to understand and design, and asks for the individual's conscious attitudes.
Disadvantages - influence of words on response chosen (eg: "very strongly disagree" rarely chosen) and middle point ambiguity (ie: what does choice of "unsure" mean?).

[18] Confirmatory factor analysis is a form of replication of the original results with a different sample.

[19] This is a measure of external reliability or consistency of the test. The scores of the same individual completing the test at two different points in time are correlated. Significant correlations are taken as evidence of reliability of the questionnaire.

self-esteem. While high Guilt scores correlated with anxiety, depression, obsession, and disordered eating behaviour, and low self-esteem (table 9.3).

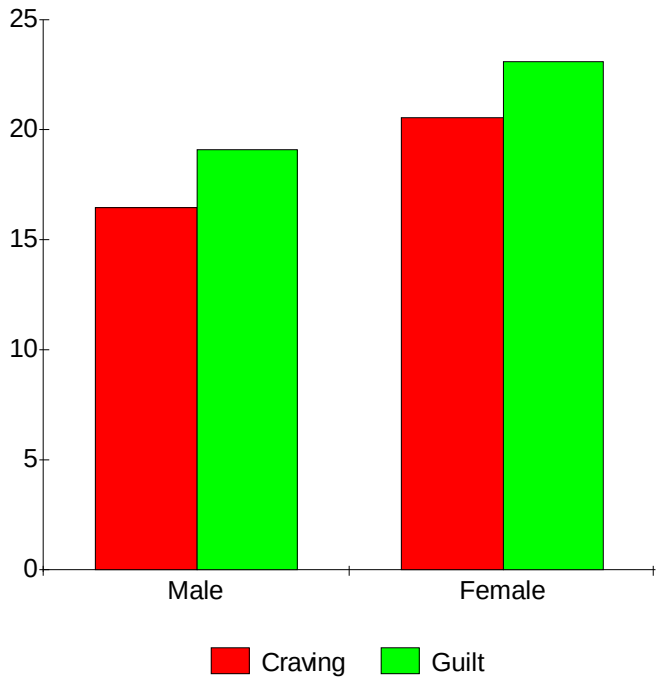

Figure 9.2 - Mean scores on two items as found by Cramer and Hartleib (2001).

BEHAVIOUR	MEASURED BY	CRAVING	GUILT
Anxiety	Spielberger State-Trait Anxiety Inventory 6-item short-form (Marteau & Bekker 1992)	ns	0.28 **
Depression	Centre for Epidemiologic Studies of Depression Scale (Radloff 1977)	0.21 *	0.24 *
Disordered eating	Eating Attitudes Test (EAT-26) (Garner et al 1983)	ns	0.53 ****
Obsession	Obsessiveness scale from MMPI-2 (Butcher et al 1989)	0.39 ****	0.32 **
Self-esteem	Rosenberg Self-Esteem Scale (Rosenberg 1965)	-0.34 ***	-0.31 **

(ns = not significant; * p<0.05; ** p<0.01; *** p<0.001; **** p<0.0001)

(After Cramer and Hartleib 2001)

Table 9.3 - Significant correlations between Craving and Guilt and other behaviours.

Müller et al (2008) produced a German version of the ACQ using two samples - 231 healthy adults and 106

students. Factor analysis produced two clear factors - Craving and Guilt. Female respondents again scored significantly higher on these two aspects of the ACQ.

Validity [20] of the questionnaire was established by correlating the Craving score, for example, with the self-reported amount of chocolate consumed per week ($r = 0.66$; $p<0.001$).

Cartwright and Stritzke (2008) built on the ACQ with a measure of chocolate craving - Orientation to Chocolate Questionnaire (OCQ) - which had three underlying factors: approach (6 items), avoidance (2 items), and guilt (6 items). There are fourteen statements with a Likert-type response choice varying from 1 ("not at all") to 9 ("very strongly") (table 9.4).

UNDERLYING FACTOR	EXAMPLE OF STATEMENT
Guilt	"After eating chocolate I often wished I hadn't"
Approach	"I was thinking about chocolate a lot of the time"
Avoidance	"I did things to take my mind off chocolate"

Table 9.4 - Examples of statements on the OCQ.

REFERENCES

Benton, D et al (1998) The development of the Attitudes to Chocolate Questionnaire Personality and Individual Differences 24, 4, 513-520

Butcher, J.N et al (1989) Development and Use of the MMPI-2 Content Scales Minneapolis: University of Minnesota Press

Cartwright, F & Stritzke, W.G.K (2008) A multidimensional ambivalence model of chocolate craving: Construct validity and associations with chocolate consumption and disordered eating Eating Behaviours 9, 1-12

Cramer, K.M & Hartleib, M (2001) The Attitudes to Chocolate Questionnaire: A psychometric evaluation Personality and Individual Differences 31, 931-942

Garner, D.M et al (1983) Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia International Journal of Eating Disorders 2, 15-34

Likert, R.A (1932) A technique for the measurement of attitudes Archives of Psychology 140, p55

Marteau, T.M & Bekker, H (1992) The development of a six-item short-form of the State Scale of the Spielberger State-Trait Anxiety Inventory (STAI) British Journal of Clinical Psychology 31, 301-306

Müller, J et al (2008) The Attitudes to Chocolate Questionnaire:

[20] Validity is related to whether the questionnaire measures what it claims to measure. A reported craving for chocolate should manifest as actually eating large amounts of chocolate if the questionnaire is valid.

Psychometric properties and relationship to dimensions of eating Appetite 50, 499-505

Osgood, C.E et al (1957) The Measurement of Meaning Urbana: University of Illinois

Parker, G & Crawford, J (2007) Chocolate craving when depressed: A personality marker British Journal of Psychiatry 191, 351-352

Radloff, L.S (1977) The CES-D scale: A self report depression scale for research in the general population Applied Psychological Measurement 1, 385-401

Rosenberg, M (1965) Society and the Adolescent Self-Image Princeton, NJ: Princeton University Press

10. USE OF SYSTEMATIC LITERATURE REVIEWS: ANTI-DEPRESSANTS

When there are many studies on a particular topic, a literature review can help practitioners, in particular, understand the main conclusions, especially when studies are contradictory (table 10.1). Systematic literature reviews are performed by researchers as part of the Cochrane Collaborative Project [21]. The emphasis is upon systematic comparison of studies, especially in relation to quality, using clear criteria. These criteria are outlined with the example of a review of the use of anti-depressants with a sleep disorder (Vignatelli et al 2008).

STRENGTHS	WEAKNESSES
1. Helps to clarify large number of studies, particularly if contradictory. 2. Convenient summary for practitioners. 3. Systematic comparison for studies based on methodological quality.	1. Many studies are not comparable using different research methodologies and designs. 2. Does not attempt to standardise findings from different studies as meta-analysis does statistically. 3. Can depend upon subjective opinion of reviewers.

Table 10.1 - Main strengths and weaknesses of systematic literature reviews.

Criteria for Reviews

1. Objectives of a review.

a) To see if anti-depressants are more effective than placebo/no treatment with aspects of narcolepsy.

b) To see if one class of anti-depressants is better than another with aspects of narcolepsy.

2. Criteria for considering studies for review.

i) Types of studies - studies comparing a treatment and non-treatment group, or comparing two different treatments.

[21] This is a database of systematic reviews of treatments (http://www.cochrane.org/index.htm).

ii) Types of participants - adults (18 years and above) with narcolepsy.

iii) Types of interventions - any dose from four chemical types of anti-depressants: tricyclics, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, and other.

iv) Types of outcome measures - eg: reduction of daytime sleepiness; quality of life; side effects; drop-outs.

3. Search for relevant articles - eg: Cochrane Central Register of Controlled Trials (CENTRAL), PsycINFO database.

4. Assessment of methodological quality.

i) Random selection and allocation of participants - adequate, inadequate, unclear.

ii) Blinding (participants and administrators of treatment; ie: not aware of group) - adequate, inadequate, unclear.

iii) Diagnostic criteria used to assess narcolepsy.

The authors concluded their review that there was "no good quality evidence that anti-depressants are effective for narcolepsy or improve quality of life" (Vignatelli et al 2008 p2).

SMALL SCALE EXAMPLE OF LITERATURE REVIEW

For the purposes of an example, the words "trial, depression, adolescents, anti-depressants" were searched in Google Scholar [22]. The first two articles that described clinical trials of anti-depressants with adolescents with depression that were freely available to download the whole article were chosen. Obviously, this is a biased process (based on convenience) whereas a full literature review would find all the relevant studies using multiple databases.

The two studies found in order were:

- Wagner et al (2004) - A comparison of citalopram and a placebo with children and adolescents with major depressive disorder. 138 patients, recruited from 21 US

[22] At http://scholar.google.com/ on 23/11/09.

sites, completed the eight-week study (71 in the treatment group and 67 in the placebo group).

- Wagner et al (2003) - A comparison of sertraline and a placebo with children and adolescents with major depressive disorder. 299 patients completed the ten-week study (143 in the drug group and 156 in the placebo group). They were recruited from fifty-three sites in the USA, India, Canada, Costa Rica, and Mexico.

1. Objectives of review - To see if selective serotonin reuptake inhibitor (SSRI) anti-depressants are more effective than placebo for adolescents with major depressive disorder.

2. Criteria for considering studies for review.

i) Type of studies - randomised, double-blinded placeb-controlled trials.

ii) Type of participants - see table 10.2.

iii) Type of interventions - see table 10.2.

iv) Types of outcome measure - see table 10.2.

3. Search for relevant articles - see above (Google Scholar).

4. Assessment of methodological quality - see table 10.2.

Based on the two studies in this example of a literature review, SSRI anti-depressant use was found to significantly reduce symptoms of major depression over a period of approximately two months compared to a placebo.

CRITERIA	WAGNER ET AL (2004)	WAGNER ET AL (2003)
Length of study	8 weeks (plus 1 week lead-in)	10 weeks (plus 2 week lead-in)
Type of participants	7-11 and 12-17 year-olds with major depressive disorder	6-11 and 12-17 year-olds with major depressive disorder
Type of interventions	Citalopram: 20mg per day initially with option to increase to 40mg per day after week 4	Sertraline: 25mg or 50mg per day; dose could be increased every 2 weeks to maximum of 200mg per day
Types of outcome measures	CDRS-R: score of 28 or less	CDRS-R: adjusted total score reduction of 40% (ie: -17 points)
Diagnostic criteria	DSM-IV criteria for major depressive disorder with current episode at least 4 weeks in duration; excluding co-morbidity, suicide risk, other anti-depressant or medication; score of at least 40 on CDRS-R	DSM-IV criteria for major depressive disorder with current episode at least 6 weeks duration; excluding co-morbidity, suicide risk, previous sertraline trial and/or failure to respond, other medication, receiving cognitive-behavioural therapy, any therapy for depression; CDRS-R score of at least 45
Random selection and allocation of participants	Adequate	Adequate
Blinding	Adequate	Adequate
Results	At week 8: 36% citalopram group vs 24% placebo group achieved response criteria (significant difference)	At week 10: 69% sertraline group vs 59% placebo group achieved response criteria (significant difference)

(CDRS-R = Children's Depression Rating Scale - Revised [23]).

Table 10.2 - Review criteria and two studies involved.

REFERENCES

Poznanski, E.O & Mokros, H.B (1996) Manual: Children's Depression Rating Scale - Revised Los Angeles: Western Psychological Services

Vignatelli, L et al (2008) Anti-depressant drugs for narcolepsy Cochrane Database of Systematic Reviews 1, Art No: CD003724

Wagner, K.D et al (2003) Efficacy of sertraline in the treatment of

[23] CDRS-R (Poznanski and Mokros 1996) has 17 items rated by clinician measuring severity of depression. Possible scores range from 17 to 113. Fourteen of the items rated 1-7 with 3 as mild, 4-5 moderate, and 7 severe symptoms. The other items rated 1-5 (Wagner et al 2003).

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children and adolescents with major depressive disorder: Two randomised controlled trials Journal of the American Medical Association 290, 8, 1033-1041

Wagner, K.D et al (2004) A randomised, placebo-controlled trial of citalopram for the treatment of major depression in children and adolescents American Journal of Psychiatry 161, 1079-1083

11. THIS HOUSE BELIEVES THAT PSYCHOANALYSIS DOES NOT WORK

FOR

1. In 1952, Hans Eysenck produced a detailed evaluation of studies of the effectiveness of psychoanalysis. It included over 7000 cases. Only 44% of individuals undergoing psychoanalysis were rated as cured, much improved, or improved. This compared with 66% of people who got better without any treatment (spontaneous remission), and 64% who improved with other therapies.

2. Psychoanalysis is based upon a number of theoretical constructs, which are difficult to prove, like the unconscious, defence mechanisms, and the Oedipus complex.
It is impossible to prove these ideas scientifically: "So if analysts see castration anxiety in their patients, Freud is right; and if they fail to see it, they have 'overlooked it' - and Freud is still right" (Tavris and Wade 1995 p452).

3. The answer to any problem (varying from phobia to psychosis) in psychoanalysis is to search for hidden explanations in the unconscious and early childhood. This does not help individuals who need solutions quickly. Other therapies, like cognitive-behavioural therapy (CBT), concentrate upon the individual's thinking patterns here and now, and relate directly to the problem.

4. Traditional psychoanalysis is recommended five times a week for at least three years. This is very expensive and time-consuming with no guarantee of improvement.

5. There are many different types of psychoanalysis today - Freudian, Jungian, Kleinian (to name but three). How to know which one to use as they are all slightly different in their emphasis?

AGAINST

1. Eysenck's analysis of the data in his 1952 study was biased against psychoanalysis. If individuals who drop-out from treatment are removed, 66% of individuals benefit from psychoanalysis. Furthermore, if the criteria for improvement are changed, 83% of people benefit from psychoanalysis and only 30% get better without any treatment (Bergin 1971).

2. Psychoanalysis is based upon a complex set of ideas

because the human being is complex. It pays attention to internal conflicts and hidden motives. Behaviour therapy, for example, treats the individual as a product of past conditioning, and sees their behaviour as no different to that of animals.

3. Psychoanalysis is the only therapy that allows a long-term study of the individual's personality. Too many therapies claim a quick solution. Human problems have origins that a "quick-fix" will not permanently solve.

4. For those who cannot afford traditional psychoanalysis, modern versions like brief focal therapy concentrate upon a particular issue for a limited time (eg: once a week for six weeks).

5. Psychoanalysis is based upon over one hundred years of ideas, originally from Sigmund Freud, but then from many others. Vast amounts have been written about psychoanalysis. Some modern therapies are only a few years old.

REFERENCES

Bergin, A.E (1971) The evaluation of therapeutic outcomes. In Bergin, A.E & Garfields, S.L (eds) Handbook of Psychotherapy and Behaviour Change: An Empirical Evaluation (2nd ed) New York: Wiley

Eysenck, H.J (1952) The effects of psychotherapy: An evaluation Journal of Consulting Psychology 16, 319-324

Tavris, C & Wade, C (1995) Perspectives in Psychology New York: Longman

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12. WEB SEARCH TASKS

1. Bipolar Disorder and Creativity

It is believed by many people that madness and creativity go together. Look for examples of famous writers and artists who suffered from bipolar disorder, and examples of those who did not (http://www.neuroticpoets.com).

Details of research (http://tinyurl.com/6a9csw), (http://tinyurl.com/5z4kwz).

2. Mental Illness and Famous Historical Figures

Joan of Arc claimed to hear the voice of God speaking to her. Today she may be diagnosed with schizophrenia for such a claim (http://www.stjoan-center.com/topics/jgrundy.html).

Find details of historical figures including political leaders and the mental illnesses they may have suffered with, eg: (http://tinyurl.com/6bpf35).

See research (http://tinyurl.com/5qqpjs).

13. DELIBERATE SELF HARM IN TWO DIFFERENT AGE GROUPS

Non-fatal deliberate self-harm (DSH) is not necessarily a "failed" suicide attempt, but is a specific desire to hurt the self, though it can provide insight into suicide (Dennis et al 2005). Characteristics of the behaviour will vary between different groups in society.

OLDER ADULTS

Dennis et al (2005) compared seventy-six adults with depression aged 65 years and over who had self-harmed with fifty similarly aged depressive controls in Leicestershire, England. All participants completed various measures of depression, and about their life situation (eg: social contacts, life events in last six months).

The two groups were similar in many ways (eg: type of depression, number of negative life events), but there were some significant differences. The DSH group reported greater feelings of hopelessness, and lower scores on the question: "Do you think it is wonderful to be alive now?" [1].

Also they had more thoughts about suicide and DSH, and reported being more sad yet crying less [2]. Furthermore, the self-harmers had poorly integrated social networks (eg: loneliness, isolation from others) (76% of DSH group vs 52% of controls) [3].

Scores on the rating instruments produced ordinal data, so statistical analysis used the Mann-Whitney U test (appendix 13A).

ADOLESCENTS AND YOUNG ADULTS

Mental Health Foundation (2006) noted the problems in gaining accurate figures for DSH among adolescents, like a failure to seek treatment for injuries. Estimated figures varied from 1 in 10 to 1 in 5 teenagers in the UK.

In terms of research, Hawton et al (2002) used a representative sample of 15-16 year-olds in schools in the Midlands. In the previous year 6.9% of respondents reported an act of DSH (as clearly defined by researchers), which was 11.2% of girls and 3.2% of boys.

---

[1] Measured on Geriatric Depression Scale (GDS-15) (Sheikh and Yesavage 1986).
[2] Measured on Beck Depression Inventory (BDI) (Beck and Steer 1993).
[3] Measured on Social Contact Schedule (SCS) (Dennis et al 2005).

Young et al (2007) concentrated on 18-20 year-olds in urban West of Scotland, and the differences in prevalence in DSH based on gender, parental social class, and current labour market position (table 13.1).

CRITERIA	DEFINITION/CATEGORIES
Parental social class	Occupation of head of household: father figure's current or previous occupation if not working or, in his absence, mother figure's current or previous occupation
Current labour market position	Full-time education (higher or further education), training or work (either full- or part-time, or on a training course or scheme), non-labour market (unemployed, at home or with care responsibilities, and sick or ill)

Table 13.1 - Definition of criteria used.

The lifetime prevalence of DSH was 7.1% with 1.6% current (ie: in past year). Women were more likely to have ever self-harmed, and individuals not working were significantly more at risk (table 13.2). Parental social class was not a risk factor.

	FEMALE (n = 618)	MALE (n = 640)	FULL-TIME EDUCATION (n = 659)	WORK (n = 464)	NOT WORKING (n = 135)
Current DSH (%)	1.9	1.3	0.9	1.1	6.7
Ever DSH (%)	8.4	5.8	5.3	6.7	17.0
Most common method (%)	Cutting any part of body (5.7)	Cutting any part of body (2.7)	Cutting any part of body (3.9)	Taking dangerous pills (3.4)	Cutting any part/ pills (10.4)
Main reason given for DSH (%)	Relieve anger/ to forget something (46.2)	Relieve anger (59.5)	Relieve anger (57.1)	Relieve anger (51.6)	Relieve anger/ to kill myself (43.5)
Main reason for ceasing (%)	Realised harm (30.0)	Realised harm (48.0)	"One off" (42.9)	Coped or felt better (44.0)	Realised harm (58.3)

(After Young et al 2007)

Table 13.2 - Summary of main gender and labour market position differences.

In terms of treatment, Livesey (2009) reported the success of a policy of offering alternative coping

techniques like the use of ice, rubber bands and marker pens instead of sharp objects; diaries; relaxation and distraction at Oakwood Young People's Centre, Sheffield, England. Episodes of DSH dropped from a peak of 8.1 per week to 0.2 after implementation of the policy.

APPENDIX 13A - MANN-WHITNEY U TEST

The Mann-Whitney test is presented as a non-parametric alternative to the t-test. Whereas the t-test compares means, the Mann-Whitney test compares medians. But the Mann-Whitney test can also detect differences in the spread of data when the medians are similar (Hart 2001). So it is important not just to report a P value, "differences in spread may sometimes be as clinically important as differences in medians, and these need to be made clear to the reader" (Hart 2001 p393).

Take the example of the hypothetical data in table 13.3 - test scores of two groups. The median for group A is three, and for group B four. There is not much difference in terms of average, and the Mann-Whitney test is non-significant. But the data are more skewed in group B, and there is a bigger difference between the scores of the fourth and the fifth participants (9 and 10 in group B) in each group.

PARTICIPANT	GROUP A SCORES	PARTICIPANT	GROUP B SCORES
1	1	6	1
2	2	7	2
3	3	8	4
4	4	9	6
5	5	10	10
Median	**5**	Median	**4**

Table 13.3 - Hypothetical scores on test.

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Beck, A & Steer, R (1993) Beck Depression Inventory Manual San Antonio, TX: Psychological Corp

Dennis, M et al (2005) Self-harm in older people with depression British Journal of Psychiatry 186, 538-539

Hart, A (2001) Mann-Whitney test is not just a test of medians: Difference in spread can be important British Medical Journal 323, 391-393

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Bulletin 33, 10-12

Mental Health Foundation (2006) Truth Hurts London: Mental Health Foundation

Sheikh, J & Yesavage, J (1986) Geriatric Depression Scale: Recent evidence and development of a shorter version. In Brink, T.L (ed) Clinical Gerontology: A Guide to Assessment and Intervention New York: Haworth

Young, R et al (2007) Young people who self-harm British Journal of Psychiatry 191, 44-49

14. RESEARCHING PRE-NATAL INFLUENCES ON ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

What happens in the womb can have an affect on later development as shown by, for example, exposure to high levels of alcohol and foetal alcohol syndrome, or the thalidomide drug and physical abnormalities (box 14.1).

> Anti-depressants and depression are both a problem for unborns. The use of selective serotonin reuptake inhibitor (SSRI) anti-depressants during early pregnancy has been linked to a heart defect in the child (Pedersen et al 2009). However, maternal depression during pregnancy has led to children who develop later in terms of smiling, talking and motor skills (Deave et al 2008).
>
> In this difficult situation of problems either way, the American College of Obstetricians and Gynaecologists and the American Psychiatric Association in August 2009 recommended that pregnant women with depression be treated with psychotherapy (Wilson 2009).

Box 14.1 - Depression and anti-depressants during pregnancy.

Recently, pre-natal causes of ADHD have been reported by studies - maternal smoking in pregnancy, undernutrition and consequent low birth weight, and gestational stress (stress experienced by the mother during pregnancy) (Thapar and Rutter 2009). Each factor causing physiological changes in the womb.

In order to establish the causal link the experimental method is best. Individuals are randomly allocated into an experimental (eg: smoking) or a control (eg: not smoking) condition. Such deliberate manipulation is, of course, unethical if not impossible.

This means that researchers must rely on naturally-occurring events to study - natural experiments (quasi-experimental method) (table 14.1). In the example of maternal smoking, it means making use of the fact that some mothers smoke during pregnancy. Huizink and Mulder (2006) reported data collected with quasi-experimental methods that support the increased risk of ADHD with maternal smoking in pregnancy.

ADVANTAGES
• Studying the effects of natural events
• Clues to cause and effect
• Some things not possible to control
• Some things not possible to study in an experiment
• Not unethical compared to the laboratory experiment equivalent
DISADVANTAGES
• Little information on participants
• Lack of control of confounding variables
• No random allocation of participants to groups
• No baseline measure of behaviour
• Not replicable usually

Table 14.1 - Advantages and disadvantages of the natural experiment method.

But how to distinguish pre-natal from post-natal effects? Mothers who smoke during pregnancy will probably also smoke during the child's early life. Also how to control for genetic inheritance (Thapar and Rutter 2009)?

The latter can be addressed in different ways:

1. Studies of children of twins - eg: Knopik et al (2006).

Knopik et al (2006) studied 268 pairs of Australian twin mothers with children aged 13-21 years. The focus was the mother's alcohol abuse and the offspring's development of ADHD. The link found between parental alcohol use and offspring ADHD could occur in three ways:

- Effects of alcohol pre-natally - teratogenic effects of alcohol on foetus development which produces physiological changes and later ADHD behaviour;
- The environment after birth - behaviours by the parents, like being emotional unavailable, or family conflict and stress during the child's life;
- Genetic transmission - eg: mothers who drink during pregnancy may themselves have ADHD, or there are common genes between ADHD and alcohol abuse (Knopik et al

2006).

The children-of-twins design offers a way to disentangle environmental and genetic factors, particularly with identical (monozygotic, MZ) twins:

a) One twin has disorder and other does not. If offspring of non-disorder twin develops the disorder, genetics must be important as the environment was different.

b) If offspring of non-disordered twin does not develop disorder, but offspring of disordered twin does, this is seen as due more to environment (figure 14.1).

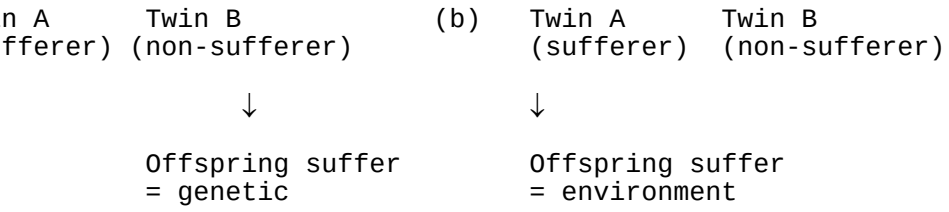

Figure 14.1 - Predictions from children-of-twins design.

Knopik et al divided the participants into groups for analysis:

- (i) Both twin mothers have alcohol problems - high genetic and high environment risk for offspring;
- (ii) One twin alcoholic and other not - high genetic and low environment risk;
- (iii) Both twins unaffected - low genetic and low environment risk.

The risk of ADHD was found to be greater in group (i) and (ii) than (iii) above. The rate of ADHD among offspring of non-sufferer twins (11.9%) would suggest that genetic factors are more important than environmental ones (table 14.2).

GROUP	RISK	PREVALENCE OF ADHD (%)
Both twins affected	High G/high E	9.2 - 10.1 *
Mother unaffected/MZ twin unaffected	High G/low E	11.9
Mother unaffected/DZ twin unaffected	Intermediate G/low E	1.6
Both unaffected	Low G/low E	4.8

(* Depends if alcohol disorder or alcohol abuse; G = genetic; E = environment; DZ = dizygotic (non-identical))

(After Knopik et al 2006)

Table 14.2 - Rates of ADHD among offspring of twins with or without alcohol problems.

2. Surrogacy.

A foetus that is genetically unrelated to the pregnant woman when implanted by assisted reproductive technology (eg: Rice et al 2009).

Rice et al (2009) noted that "the difficulty of disentangling pre-natal effects from inherited influences is not easily solved except through experimental methods that to date have only been possible in animal studies" (p2464).

In vitro fertilisation (IVF) allows for a number of possibilities - child related to both parents (homologous IVF - conception in laboratory and implanted in womb), to mother only (IVF with sperm donation), the father only (IVF with egg donation), or to neither parent (IVF with embryo donation) (Rice et al 2009). IVF allows researchers to try and disentangle genetic and environmental factors (table 14.3).

Homologous IVF	IVF with sperm donation	IVF with egg donation	IVF with embryo donation
G and E	Mother G, and E	Father G, and E	E

(G = common genetic link between parent and offspring; E = common environment between parent and offspring)

Table 14.3 - Types of IVF and genetic and environmental factors.

Rice et al (2009) studied 779 children born between 1994 and 2002 through IVF who were related to the mother (homologous, sperm donation) or not (embryo donation) who provided the pre-natal environment. Maternal smoking

during pregnancy was self-reported, and child's anti-social behaviour was reported by mother and father.

Two key findings were noted:

a) Mothers who smoked during pregnancy (6% of women) had significantly lower birth weight offspring (mean: 2,771g vs 3,091g) irrelevant of genetic relationship between mother and foetus. This confirmed that prenatal smoking has a measurable effect on the foetus.

b) Mothers who smoked had offspring with higher anti-social behaviour, but there was only a significant association in the genetically related group (table 14.4). This highlighted the role of genetic factors (as well as environmental) in explaining the child's anti-social behaviour.

	MOTHER SMOKES	DOES NOT SMOKE
MOTHER-FOETUS RELATED	2.54	1.75 *
UNRELATED	1.77	2.08

(* Significant difference at p<0.001 for that row)

Table 14.4 - Mean scores of children's anti-social behaviour on maternal smoking and genetic relatedness.

3. Siblings exposed to different pre-natal environments - ie: maternal smoking in one case and not the other (eg: D'Onofrio et al 2008).

D'Onofrio et al (2008) used data from the US National Longitudinal Survey of Youth (NLSY). In terms of rates of ADHD and other problem behaviours, offspring were compared to their siblings based on difference in exposure to maternal smoking during pregnancy (table 14.5). There were no differences in rates of these behaviours which is opposite to predicted if pre-natal smoking caused them. If smoking in pregnancy caused ADHD, it would be expected that rates in sibling 1 should be higher than sibling 2 in experimental group A and opposite in experimental group B.

	CONTROL A	CONTROL B	EXPERIMENTAL A	EXPERIMENTAL B
Sibling 1	Smokes	Does not	Smokes	Does not
Sibling 2	Smokes	Does not	Does not	Smokes

Table 14.5 - Design of study groups in relation to maternal smoking during pregnancy.

Thapar and Rutter (2009) concluded that: When taken together, all these findings across different designs suggest that the claim that maternal smoking in pregnancy causes ADHD in offspring may be unfounded or the effects may be much smaller than currently believed" (p101).

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15. EVOLUTION AND DEPRESSION

Depression is a pathological behaviour for an individual because, at the extreme, it may lead to suicide. To a lesser degree, it can involve lack of motivation to eat, and a loss of libido. So in terms of evolution, depression does not appear to be beneficial to the individual, when the purpose is to survive and pass on genes to the next generation.

But evolutionary explanations are not just about behaviours directly related to survival (ie: avoid predators, find food, and mate), there are also indirectly related behaviours. This is so when animals live in groups. Certain behaviours related to harmony in the group and other group behaviours become important in terms of evolutionary adaptation. Early humans lived in such groups, and behaviours that evolved to aid this will remain with us today.

A key aspect of group living is conflict about resources between members of the group. Among many group living animals social rank and hierarchy has evolved to avoid endless (wasteful) conflicts. Thus the dominant animal will also have first access to food and mates, and subordinates have to accept this order.

The evolution of depression in humans has been linked to this process by social rank theory (Price 1972). In a conflict situation, one individual attempts an "involuntary subordinate strategy" (ISS) to signal that they are no longer a threat and to save themselves from (further) injury. The behaviours associated with depression evolved as an ISS and to aid group harmony (Sturman and Mongrain 2005).

"By understanding the functions of ISS we can account for the biological state of depression, the psychological preoccupations (self as inferior, worthless, and powerless), the anxieties (major losses and separation), the social behaviour (inhibited, retarded) and the triggers of depression (major losses of allies and friends that reduce self-esteem and confidence, loss of status, and devaluing, rejecting environments)" (Sloman et al 1994 pp404-405 quoted in Sturman and Mongrain 2005 p506).

If depression has evolved for such purposes, two aspects of personality with be crucial (Sturman and Mongrain 2005):

- Social comparison - individuals will be making comparisons of themselves with others to check who is more dominant and who they must be submissive towards. This produces a heightened awareness of those better (causing low self-esteem) which is a symptom of

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depression.

- Entrapment and arrested flight - when animals cannot escape, "arrested flight" occurs, which is behaviour that signals no threat to others (eg: immobile, turn head away). The aim is to stop or limit further attack from the dominant animal. Arrested flight has been described in humans as "entrapment" (Gilbert and Allan 1998). The individual feels trapped both literally by external circumstances (eg: cannot leave job) or metaphorically by their own thoughts (eg: cannot stop thinking about something), whether they are actually trapped. These are behaviours again related to depression.

These two aspects of personality are manifest in self-criticism. Sturman and Mongrain (2005) investigated these behaviours among students in Toronto, Canada who had experienced at least one prior episode of major depression. Questionnaires were completed to measure social comparison, entrapment, self-criticism, and depression. These measures were found to correlate together.

Self-criticism leads to negative social comparisons (ie: feeling inferior to others) and internal entrapment (not being able to escape self-critical thoughts), and further on to depression. What may have evolved as an adaptive behaviour in early humans can be seen as unhelpful in individuals today.

Another view is that depression evolved as a way to reduce the motivation when the probability of gaining a reward is low (eg: Klinger 1975; incentive disengagement). Otherwise, continuing to pursue such rewards would be energy expensive with little pay-off.

The evolution of depression, or more specifically, despair can be linked to attachment. When young are separated from their mother, they protest, usually through calling. But such calling is risky as predators may be attracted. Despair evolved to keep the young animal quiet and immobile which is the best strategy for survival until the mother returns. The genes involved in evolution carry advantages in some environments, but also risks in other (Gilbert 2006).

Gilbert (2006) preferred social origins to the evolution of depression in humans (eg: submissiveness) rather than non-social theories, like incentive disengagement.

These evolutionary explanation are speculative because there is no way of knowing that the behaviours existed or how they existed in early human groups.

Behaviours like these do not leave fossil evidence.

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16. FOUR MYTHS ABOUT TOURETTE'S DISORDER

DSM-IV-TR (APA 2000) emphasised the presence of tics in diagnosing Tourette's disorder (TD) (box 16.1). There are motor tics, which involve repetitive physical actions like facial grimacing or shrugging of the shoulders, phonic tics (eg: throat clearing), and series of actions (eg: picking up and smelling objects). Tics themselves are usually divided into simple and complex (table 16.1).

About 1-3 out of 1000 children develop TD by around 6-7 years old [4], with the majority being male (Lilenfeld and Arkowitz 2009). There is often co-morbidity with other conditions like obsessive-compulsive disorder or attention deficit hyperactivity disorder (box 16.2).

- Both multiple motor tics and one or more vocal tics present at the same time if not currently.
- Tics occur many times a day or nearly every day or intermittent throughout a period of more than one year.
- Onset before 18 years old.
- No other physiological cause; eg: stimulant use, Huntington's.

(After Zinner 2004)

Box 16.1 - Diagnostic criteria for TD in DSM-IV-TR.

	SIMPLE	COMPLEX
MOTOR TICS	Eye blinking Facial grimacing Neck jerking	Jumping Hand gestures Echopraxia (imitations of another's movements)
PHONIC TICS	Coughing Sniffing Clicking	Single words or phrases Palilalia (repeating own words) Speech blocking (eg: stuttering)

Table 16.1 - Examples of simple and complex tics.

Robertson and Cavanna (2007) reported the case of a ten year-old boy in the UK with TD and obsessive-compulsive disorder who believed he had caused the terrorist attacks in the USA on September 11, 2001 by failing to perform certain compulsive rituals. In particular, he had failed on that day to step correctly on a certain white mark on the road as is his ritual.

Box 16.2 - Unusual case of TD and obsessive-compulsive behaviour.

[4] However, Zinner (2004) estimated 1% of children based on US studies, while transient tics lasting less than one year occur in 20% of schoolchildren.

Lilenfeld and Arkowitz (2009) listed common myths about sufferers of TD.

1. The main symptom is the uncontrollable use of curse words (coprolalia) and obscene gestures (copropraxia).

Because of cases of TD in the media either in real-life documentaries or in humour which emphasise this symptom, the majority of the public believe this statement to be true. In reality, only a minority of sufferers show this symptom (10-15%; Lilenfeld and Arkowitz 2009).

2. The symptoms are voluntary.

Each symptom has a "premonitory urge" (powerful desire to emit the tic), which can be suppressed on occasions, but this can produce a "rebound" of tics later.

3. The cause of the disorder is an underlying psychological conflict.

Psychoanalytic explanations for TD dominated the early understanding, like the "symbolic discharge of repressed sexual energies". Modern research has shown a genetic basis (eg: 53% concordance rate among MZ twins and 8% for DZ twins; Price et al 1985), and brain abnormalities (eg: in basal ganglia area).

4. There is no treatment for TD.

Though there is no known cure, there are several treatments that have been shown to reduce symptom frequency including drugs (eg: haloperidol) and behaviour therapies (eg: habit reversal) (Lilenfeld and Arkowitz 2009).

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17. INCREASING THE AVAILABILITY OF MEDICATION

In the UK, as in many countries, the availability of medication is controlled as is the advertising for it. Over-the-counter (OTC) medication can be purchased by individuals in pharmacies, and direct-to-consumer-advertising (DCTA) is permitted (appendix 17A). Other drugs are prescription-only-medication (POM) and advertising for these is limited to medical professionals.

The changing of certain drugs from POM to OTC has important implications. It could reduce the pressure on doctors for commonly prescribed drugs. While pharmaceutical companies can "exploit" their brand names directly to the consumers (Rubin and Wylie 2009).

The drug that is the number one celebrity of brand name drugs is "Prozac" (a type of selective serotonin reuptake inhibitor anti-depressant) (appendix 17B) [5].

The brand name issue is the case with "Viagra" (brand name of sildenafil drug) licensed to use with erectile dysfunction. It has become famous and moved into the category of "lifestyle drug" - "a medicine that is used to satisfy a non-health-related goal or is used for treating problems that lie at the margins of health and well being" (Gilbert 2000). It has become a "recreational commodity" as a "recreational aphrodisiac" (Rubin and Wylie 2009).

Delate et al (2004) investigated the early use of Viagra (after its launch in 1998) in the USA. The growth of demand was mainly due to more use by younger men (ie: 18-45 years old) as opposed to the core age group traditionally diagnosed with erectile problems (40-70 years old). In fact, few users were found to have erectile dysfunction.

While, in the UK, Aldridge and Measham (1999) reported the illicit use of Viagra in nightclubs in Manchester as a cocktail with other illegal drugs, like amphetamines.

As with any market for products, competitors arrived for sildenafil in the form of vardenafil (brand name "Levitra") and tadalafil (brand name "Cialis"). The producers of Viagra, Pfizer, are thus under pressure to increase advertising and marketing for their "market leader" to maintain this position [6]. This involved DTCA in

[5] Brewer (1999) reflected on the claims and hype associated with Prozac.

[6] Brewer (2007) explored how pharmaceutical companies increased their sales in the same way as companies selling any product.

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the USA to all men for their "on and off days", and the supporting of campaigns about impotence education in other countries where DTCA is not permitted (Rubin and Wylie 2009).

Because of the pressure of demand for Viagra, Rubin and Wylie (2009) argued that it should be made available OTC in the UK with suitable screening. This would involve a full medical consultation with a pharmacist before purchase [7]. However, individuals using Viagra purely as a lifestyle drug may not wish to undergo this procedure (and cost), and will continue to purchase it elsewhere (with the associated risks of drug quality).

Making certain psychotropic drugs available OTC because the demand is high ignores the fact that pharmaceutical companies are involved in creating the demand both directly and indirectly, and the social pressures to have "a tablet for everything". Society is highly competitive and individuals will resort to any assistance to get ahead [8]. I am not sure that I like such a society, but, more importantly, the type of society that allows individuals to choose which pills they take today, irrelevant of whether they need them (appendix 17C).

APPENDIX 17A - DTCA

In the UK, DTCA is restricted for POM "in order to protect public health" (Medicines Control Agency 1999 quoted in Meek 2001). It is argued that "prescription medications are part of an intricate system of medical care that should be governed by science and careful human judgement - not the profit motive" (Meek 2001).

DTCA is permitted in the USA and New Zealand. In 2000 the New Zealand Ministry of Health reviewed the DTCA policy. On the positive side, it encouraged patients to seek beneficial help and medications, while, on the negative side, the balance of advertising was not fair and demands for certain drugs was much higher than others (Meek 2001).

Table 17.1 summarises the main arguments for and against DTCA for psychotropic drugs.

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[7] "Boot's the Chemist's" tried a pilot scheme in February 2007 to make Viagra available OTC (4 tablet pack for £50) but pharmacist supervised (ie: 1 hour consultation) (Rubin and Wylie 2009).

[8] An online survey of scientists found that 20% used prescription drugs to enhance "concentration" and 70% said that they were willing to take drugs to "boost brainpower" even with mild side effects (Haier 2009).

ARGUMENTS FOR	ARGUMENTS AGAINST
1. Encourages patients to seek beneficial treatments. 2. Aids in patient education about health and illness. 3. Pharmaceutical companies can reach people unaware of problems. 4. Part of patient empowerment. 5. Many medical professionals are not able to keep up with all the latest developments. 6. Pharmaceutical companies as competitive companies should be allowed to market their products unimpeded.	1. Advertising is not for education but profit, and so not presenting all the options. 2. Advertising focuses upon products that companies want to sell (eg: new, expensive) not necessarily the best. 3. Devalues the role and expertise of medical professionals. 4. Individuals with mental disorders may not be able to decide for themselves which drug to take. 5. Increased demand for drugs will lead to increased costs to the NHS if drugs are free or subsidised. 6. Concerns about the social construction of mental disorders to increase the market for products.

Table 17.1 - Main arguments for and against DTCA of psychotropic medications.

APPENDIX 17B - MARKETING OF ANTI-DEPRESSANTS

Disturbingly, Barber (2008) reported a study that found that 43% of Americans prescribed anti-depressants had no psychiatric diagnosis.

Barber (2008) distinguished between depression and Depression. The latter is the severe condition as captured by diagnostic classification systems, and anti-depressants were designed to combat this. The other type, depression, is "everyday blues" - "feeling down", "in the dumps" - part of the human condition, which anti-depressants are now prescribed for. Healy (2004) is very critical about this process - "a creation of depression on so extraordinary and unwarranted scale as to raise questions about whether pharmaceutical and other health care companies are more wedded to making profits from health than contributing to it" (quoted in Barber 2008 p49).

Selective serotonin reuptake inhibitor anti-depressants designed for Depression are a prime example of a drug that has increased in the disorders for which it is prescribed; eg: obsessive-compulsive disorder,

eating disorders, anxiety. This is known as "off label" prescription in the USA (ie: problems not prescribed by the Food and Drug Administration licence). It is not illegal, but lacks scientific support (Barber 2008).

APPENDIX 17C - ETHICS AND COGNITIVE ENHANCEMENT

The possibility of pharmacological cognitive enhancement of healthy individuals with few side effects is very attractive. Memory, executive functions like planning, and concentration appear susceptible to improvement (Turner and Sahakian 2006).

For example, modafinil (drug for treating sleep disorder, narcolepsy, where individuals fall asleep suddenly and often) has been found to improve short-term memory and planning abilities in healthy, young volunteers (Turner et al 2003).

Self-medication to improve everyday performance is occurring with OTC products like herbal stimulants and caffeine.

Cognitive enhancement demands ethical consideration because "so much more complex than, for example, enhancement of one's appearance, is that we primarily define and distinguish ourselves as individuals by our behaviour and personality" (Turner and Sahakian 2006 p116).

Turner and Sahakian (2006) highlighted two ethical concerns:

i) The definition of what is a "normal" healthy brain and the distinction between treatment and enhancement. If everybody is taking a particular drug, how to distinguish between those who receive it as treatment (and justify free access from the NHS) and those who use it as enhancement (and need to pay for it).

For example, two students attending an examination want a drug to help their performance. One student suffers from severe anxiety, and the other wants a high mark which leads to sleep deprivation through studying excessively. The former receives anti-anxiety drugs as treatment, while the latter wants stimulants as an enhancer. The appropriateness of giving stimulants to the latter is the ethical debate here, particularly if such behaviour as seen as "normal". In other words, "normal" functioning includes the common use of pharmacological cognitive enhancers.

ii) The consequences of widespread use of cognitive enhancers. In a competitive society, as soon as one individual or group gains an advantage, others want or feel the need to have it.

One pupil goes to their examination calm but focused because of the drugs taken, soon other parents will want their children to have the same drugs.

Also "some are concerned that if we substantially improve our overall cognitive functioning, we may alter fundamental aspects of our identity by eliminating the need to strive for success" (Turner and Sahakian 2006).

I think that is also difficult to restrict illegal drug use if "legal" drug use is acceptable. If individuals take (legal) drugs to improve concentration school, why not take (illegal) drugs to have a good time after school?

Sententia (2004), for example, argued that the use of enhancers should be part of "cognitive liberty" - the democratic right to look after your own thought processes. This does presuppose that individuals can make choices when living in a high-pressured marketing society.

So, "as long as a person's behaviour does not harm others, they should be free to take any substance they wish to, and equally, as long as a person's behaviour does not endanger others, individuals should not be compelled against their will to use technologies that will affect their cognitive liberty" (Turner and Sahakian 2006) argued people like Sententia.

But this is the extreme individualism of the West that fails to see that we are all interconnected. if nothing else, the resources for the production of cognitive enhancers for the rich (for that is who will pay for them) deprives others of those resources for something else (maybe a more basic need like survival).

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18. USE OF OFFICIAL STATISTICS: AN EXAMPLE WITH MALE SUICIDE RATES

Researchers interested in general patterns in society can make use of official statistics. These are statistics collected by government departments and organisations, and other official institutions in society (table 18.1).

STRENGTHS	WEAKNESSES
1. Governments able to collect large amounts of data, usually more than researchers can. 2. Governments can enforce responses from organisations and individuals whereas researchers are dependent on willingness to respond. 3. Governments have access to data (eg: private) that non-government bodies do not. 4. Researchers can use the data with little cost to themselves (eg: no collection costs). 5. Official statistics often exist over long periods of time allowing historical studies and comparisons. 6. Useful for general patterns in society. 7. Unobtrusive for researcher.	1. Researchers dependent on definitions of categories used in official statistics, and methods of collection. Thus it is a secondary source. 2. Official categories may miss information simply by their means of collection (eg: crime figures only based on reports to police). 3. Not all respondents may be willing to tell official bodies everything (ie: withholding of information). 4. Definitions of categories can change over time and can be different between countries making comparisons difficult. 5. Government information only (ie: only interested in data for their purposes not necessarily all cases). 6. Government statistics may be adapted for political purposes and thus not completely accurate. 7. Individual differences are lost in the general patterns.

Table 18.1 - Strengths and weaknesses of official statistics for researchers.

EXAMPLE: PRITCHARD (1992)

Pritchard (1992) used official statistics to investigate male suicide rates between 1974 and 1988. The data were used to examine three areas of interest:

1. The suicide rate of young men (15-24 years) in relation to all men in the UK.

2. Changes in male suicide rates in the UK compared to the other European Community (EC) countries over the period of the study.

3. The link between unemployment and male suicide.

The sources of the data used were:

- Annual statistics of the World Health Organisation (WHO) (1974-1990) [9] - These detail age-related standardised mortality for each country. This is usually calculated as rates per 100 000 or per million population. This standardised figure allows comparison between countries of different population sizes where absolute numbers are misleading. For example, thirty suicides in country A (population: 30 million) and country B (population: 5 million) is very different.

- Annual statistics of the International Labour Organisation (ILO) (1982-1990) [10] - These give a percentage of the workforce without paid employment for each country (appendix 18A).

- Department of Employment "Employment Gazette" (1974-1988) - These give rates of unemployment for young adults in the UK.

Findings

1. The suicide rate of young men (15-24 years) in relation to all men in the UK.

Male suicide rate for all ages showed an increase over the period of study, but the rate for young adults was a greater increase. For example, in England and Wales, the increase overall was 32%, but 90% for 15-24 year-olds, while in Scotland the increases were 68% and 163% respectively.

Problems

a) Inclusion and exclusion of death as suicide, particular if details of death unclear. For example, how to classify an individual who dies from a car crash if accident happens on a lone stretch of road? UK statistics use the WHO categories of suicide, intentional self-harm,

[9] Latest data per country at http://www.who.int/mental_health/prevention/suicide/country_reports/en/index.html.
[10] Latest data per country at http://laborsta.ilo.org/STP/guest.

and undetermined injury (ICD-10: X60-X84, Y 87.0; appendix 18B).

Take the example of poisoning, how to distinguish intention (ICD-10 X60-69) or unintentional poisoning (ICD-10 X40-49) (eg: overdose from wrong amounts of legal or illegal drugs). Determining the intention of a person who took a drug and subsequently died (ie: cannot be questioned about their motivation and behaviour) is difficult for medical professionals and misclassification may result. Some deaths are classified as suicides when unintentional and vice versa (Centers for Disease Control and Prevention 2007).

b) Establishing the age of victims.

c) Choice of age bands arbitrary - eg: why 15-24 years used for young adults?

d) With small populations (ie: those committing suicide) slight changes in the actual numbers can appear as large effects, so caution is required (Pritchard 1992).

e) Comparison between government studies is also difficult when different calculations and categories are used. The example in table 18.2 is quite straightforward as the Fitzpatrick et al figures can be multiplied by ten to make them comparable to Pritchard, but there is a difference in treating England and Wales as separate or combined.

COUNTRY	PRITCHARD (1992): per million population (1988)	FITZPATRICK ET AL (2001): per 100 000 population (1991-1997)
England & Wales	125	-
England	-	14
Wales	-	17
Scotland	173	24
Northern Ireland	156	14

Table 18.2 - Rates of overall male suicide from two sets of official statistics.

2. Changes in male suicide rates in the UK compared to the other European Community (EC) countries [11] over the

[11] Belgium, Denmark, France, Greece, Italy, Portugal, Republic of Ireland, Spain, The Netherlands, and West Germany.

period of the study.

Only Portugal and West Germany saw a decline in general male suicide rates across the period. The largest increases were in Northern Ireland (206%), Scotland (68%), Spain (67%), and Italy (62%).

For suicide among young men, only Denmark and West Germany showed declines, and the greatest increases were in Northern Ireland, Spain and Scotland.

Problems

a) Though the data were collected by the WHO, there will be country differences in the categorisation of suicide.

b) Some countries did not provide information, so the data are missing (eg: annual suicide rates for Belgium in 1980, 1981 and 1985).

3. The link between unemployment and male suicide.

Pritchard correlated the suicide rate with the unemployment rate for each country. Statistical significance of the relationship was assessed with the Spearman rank order correlation test.

There was a significant positive correlation (figure 18.1) [12] between the two variables in every country except Denmark, Portugal, and West Germany. For youth suicide, there were significant positive correlations in all countries except Denmark, Portugal, Republic of Ireland, and The Netherlands, while for West Germany the relationship was a significant negative correlation (figure 18.2) between suicide and unemployment (ie: lower unemployment leads to greater suicide).

[12] Positive correlation means that scores on both variables are similar; eg: low unemployment/low suicide rate, high unemployment/high suicide rate. Negative correlation means that scores on both variables are opposite; eg high unemployment/low suicide rate.

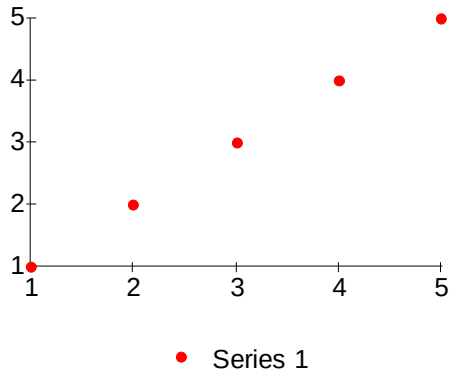

Figure 18.1 - Example of positive correlation.

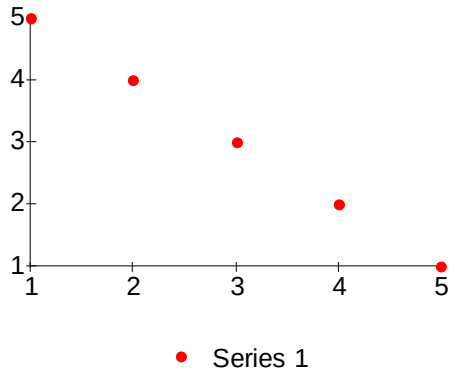

Figure 18.2 - Example of negative correlation

Problems

a) Greece, for example, did not provide unemployment data that could be used.

b) Correlations (even statistically significant) do not mean causation. So it is not possible to definitively say that unemployment causes suicide. In theory, correlation is a two-way relationship, though suicide causing unemployment is obviously impossible.

APPENDIX 18A - ILO DEFINITION OF UNEMPLOYMENT

Unemployment is defined as follows in the Resolution concerning statistics of the economically active population, employment, unemployment and underemployment, adopted by the Thirteenth International Conference of Labour Statisticians (Geneva 1982):

(1) The "unemployed" comprise all persons above a specified age who during the reference period were:

(a) "without work"; ie: were not in paid employment or self-

employment, as defined in paragraph 9;
(b) "currently available for work"; ie: were available for paid employment or self-employment during the reference period; and
(c) "seeking work"; ie: had taken specific steps in a specified reference period to seek paid employment or self-employment. The specific steps may include registration at a public or private employment exchange; application to employers; checking at worksites, farms, factory gates, market or other assembly places; placing or answering newspaper advertisements; seeking assistance of friends or relatives; looking for land, building, machinery or equipment to establish own enterprise; arranging for financial resources; applying for permits and licences, etc.

(2) In situations where the conventional means of seeking work are of limited relevance, where the labour market is largely unorganized or of limited scope, where labour absorption is, at the time, inadequate, or where the labour force is largely self-employed, the standard definition of unemployment given in subparagraph (1) above may be applied by relaxing the criterion of seeking work.

(3) In the application of the criterion of current availability for work, especially in situations covered by subparagraph (2) above, appropriate tests should be developed to suit national circumstances. Such tests may be based on notions such as present desire for work and previous work experience, willingness to take up work for wage or salary on locally prevailing terms, or readiness to undertake self-employment activity given the necessary resources and facilities.

(4) Notwithstanding the criterion of seeking work embodied in the standard definition of unemployment, persons without work and currently available for work who had made arrangements to take up paid employment or undertake self-employment activity at a date subsequent to the reference period should be considered as unemployed.

(5) Persons temporarily absent from their jobs with no formal job attachment who were currently available for work and seeking work should also be regarded as unemployed in accordance with the standard definition of unemployment. Countries may, however, depending on national circumstances and policies, prefer to relax the seeking work criterion in the case of persons temporarily laid off. In such cases, persons temporarily laid off who were not seeking work but classified as unemployed should be identified as a separate subcategory.

(6) Students, homemakers and others mainly engaged in non-economic activities during the reference period who satisfy the criteria laid down in subparagraphs (1) and (2) above should be regarded as unemployed on the same basis as other categories of unemployed identified separately, where possible.

(Source: http://laborsta.ilo.org/applv8/data/c3e.html)

APPENDIX 18B - INTENTIONAL SELF HARM (ICD-10)

In ICD-10 categories X60-X84 cover intentional self-harm, and Y87.0 includes all of the below but with a "late effect" (delayed effect) on death.

X60 Intentional self-poisoning by and exposure to nonopioid analgesics, antipyretics and antirheumatics.

X61 Intentional self-poisoning by and exposure to antiepileptic,

sedative-hypnotic, antiparkinsonism and psychotropic drugs, not elsewhere classified.

X62 Intentional self-poisoning by and exposure to narcotics and psychodysleptics (hallucinogens), not elsewhere classified.

X63 Intentional self-poisoning by and exposure to other drugs acting on the autonomic nervous system.

X64 Intentional self-poisoning by and exposure to other and unspecified drugs, medicaments and biological substances.

X65 Intentional self-poisoning by and exposure to alcohol.

X66 Intentional self-poisoning by and exposure to organic solvents and halogenated hydrocarbons and their vapours.

X67 Intentional self-poisoning by and exposure to other gases and vapours.

X68 Intentional self-poisoning by and exposure to pesticides.

X69 Intentional self-poisoning by and exposure to other and unspecified chemicals and noxious substances.

X70 Intentional self-harm by hanging, strangulation and suffocation.

X71 Intentional self-harm by drowning and submersion.

X72 Intentional self-harm by handgun discharge.

X73 Intentional self-harm by rifle, shotgun and larger firearm discharge.

X74 Intentional self-harm by other and unspecified firearm discharge.

X75 Intentional self-harm by explosive material.

X76 Intentional self-harm by smoke, fire and flames.

X77 Intentional self-harm by steam, hot vapours and hot objects.

X78 Intentional self-harm by sharp object.

X79 Intentional self-harm by blunt object.

X80 Intentional self-harm by jumping from a high place.

X81 Intentional self-harm by jumping or lying before moving object.

X82 Intentional self-harm by crashing of motor vehicle.

X83 Intentional self-harm by other specified means.

X84 Intentional self-harm by unspecified means.

(Source: http://apps.who.int/classifications/apps/icd/icd10online/).

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19. CHRONIC FATIGUE SYNDROME AND STRESS

Chronic Fatigue Syndrome (CFS) is the current name [13] for a condition of disabling fatigue involving muscle and joint pain, and changes in mood and cognitive abilities (Kato et al 2006) (box 19.1).

CFS is not included in DSM-IV, and briefly mentioned in ICD-10 (van Staden 2006). In 1994, the Centers for Disease Control and Prevention in the USA produced the following symptom list:

- Persistent unexplained chronic fatigue lasting at least six months;
- Onset is new and not the result of another disease;
- Not alleviated by rest;
- Leads to substantial reduction in occupational, educational, social and personal activities;
- At least four in last six months of: impaired memory and concentration; sore throat; tender lymph nodes; muscle pain; pain in several joints; new headaches; unrefreshing sleep; malaise after exertion (van Staden 2006).

Box 19.1 - Symptons of CFS.

Generally the knowledge about CFS is limited including precipitating factors, though stress and personality traits are involved. Kato et al (2006) investigated these factors using individuals on the Swedish Twin Registry, which contains approximately 70 000 twin births in Sweden between 1886 and 1990 [14].

CFS or CFS-like illness was established by telephone interview with questions like, "Have you felt abnormally tired during the last six months?", and the presence of symptoms like sore throat, muscle pain, and unrefreshing sleep during the period of tiredness [15].

Self-perceived stress was measured, and the Eysenck Personality Inventory (EPI) (Eysenck and Eysenck 1968) was used for personality traits [16]. The latter measures traits like introversion/extraversion and emotionally unstable (neurotic)/stable.

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[13] Other names include myalgic encephalomyelitis (ME), and "yuppie flu". However, there is some disagreement over whether CFS and ME are the same thing (Hooper 2005). Furthermore, "Many of the symptoms that people present with could be indications for other diseases, making it difficult to diagnose" (Ong et al 2005). But physiological differences in the immune system have been found among CFS sufferers which challenges the negative view of "all in the mind" that some take about CFS (Hooper 2005).

[14] Details in Lichtenstein et al (2002). It is divided into three cohorts - born 1886-1925 (details collected from all parishes in Sweden), born 1926-1958 (using nationalised birth registrations), and born 1959-1990 (fuller information).

[15] The telephone interviews took place between 1998-2002.

[16] These measures were via mailed questionnaires in 1972-3.

Of 19 192 respondents (born between 1935-1958) (out of 41 499 on register at time of study), 1570 individuals were rated as "chronic fatigue" based on their answers. Among this group, self-reported daily stress was significantly higher than controls, and there were higher scores on emotional instability (low self-esteem, anxious, depressed, guilt feelings) on the EPI.

This was a large-scale study, but diagnosis of CFS was imprecise and stress level was self-reported. For practical reasons, the different measures were not collected at the same time.

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20. DRUG TREATMENT VERSUS DRUG TREATMENT AND THERAPY FOR ADOLESCENT DEPRESSION IN THE UK

Many clinical trials and studies compare a treatment to no treatment to assess the benefits of the treatment. However, some studies have shown that a combination of treatments is better than a single treatment or no treatment.

In the case of depression, the Treatment of Adolescents with Depression Study (TADS) (March et al 2004) in the USA showed that fluoxetine (selective serotonin reuptake inhibitor; SSRI) and cognitive behavioural therapy (CBT) were beter than fluoxetine alone in reducing depression and suicidality. But the generalisability of this study to the UK has been questioned (table 20.1) (Goodyer et al 2007).

• Exclusion of adolescents with suicidal intent, self-harm, thought disorder, severe conduct disorders, and substance misuse.
• Many participants recruited by advertisement, which is associated with better response to treatment (Bimaher et al 2000).

Table 20.1 - Key problems with TADS.

The Adolescent Depression Anti-depressant and Psychotherapy Trial (ADAPT) (Goodyer et al 2007) [17] in England was designed to take account of the above weaknesses. Participants aged 11-17 with major depression were recruited from child and adolescent mental health services (CAMHS) (6 outpatient clinics) in Manchester and Cambridge. It included individuals with active suicidal intent, self-harm, depressive psychosis, and conduct disorders, but excluded individuals with schizophrenia or bipolar disorder, learning disabilities, allergy to SSRIs, and previously non-responsive to SSRI and CBT [18].

510 adolescents were initially approached to participate, and 208 individuals began the study [19] [20]. A

[17] The authors called it a "pragmatic randomised controlled superiority trial".

[18] Studies tend to exclude a number of individuals in order to give a similar sample of people. This reduces the risk of extraneous variables like other disorder. On the other hand, the success of the trial could be questioned because of the "cherry-picking" of "best" participants.

[19] The majority of participants were female - 154 (74%). Ideally, the sample would be equal numbers of males and females, but, in practice, the study was dependent on who was available through the CAMHS and who agreed to participate.

[20] The sample had the typical characteristics of adolescents with major depression in England. Only individuals with persistent depression were included. Any adolescents who responded to brief therapy

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score of seven or more on the Health of the Nation Outcome Scales for Children and Adolescents (HoNOSCA) (Gowers et al 1999) [21] was used as the baseline for inclusion. Improvements on this scale were the outcome measure at six, twelve, and 28 weeks [22].

Participants were randomly divided into SSRI alone or SSRI and CBT [23]. There was no control group with no treatment nor a placebo group nor placebo plus CBT group [24]. The SSRI used was fluoxetine with a daily dose of 10mg, which could be increased as required during the twelve weeks of the trial and the following sixteen weeks maintenance phase (mean dose given was 30mg) [25].

CBT was given weekly for twelve weeks and then fortnightly for another twelve weeks with a final session on week 28 (total sessions = 19). The CBT involved techniques like goal setting, self-monitoring of thoughts, challenging negative thinking, and problem-solving skills (appendix 20A).

In terms of the findings, both groups showed improvements over the 28 weeks of the study, and neither group was significantly better than the other (table 20.2).

	SSRI ALONE	SSRI AND CBT
Baseline	25.5 *	25.1
Week 28	14.5	15.4
Improvement	-11.0	-9.7

(* Higher score = more severe depression, thus a decline means an improvement)

(After Goodyer et al 2007)

Table 20.2 - Mean scores on HoNOSCA.

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before the study were excluded.

[21] HoNOSCA is an interviewer-rated measure based on response of participant to questions.

[22] But still only studied short-term effects of treatment.

[23] The study could not be fully blinded. Blinding is the process where the participants do not know which group they are (single blind) nor do the researchers (double blind). One group is receiving CBT as well as the SSRIs, and this attention may be a placebo effect to produce improvement irrelevant of the techniques of CBT.

[24] Control groups allow a comparison. They show what happens when there is no treatment - ie: who spontaneously gets better or improves because of the attention of being in a study. This is important because any improvement in the treatment group has to be compared to improvements in the control group. On the other hand, control groups can be seen as wasteful with participants as well as the ethics of deliberately not treating some individuals. This was the defence used by the authors. However, the ethical question can be overcome by using individuals on a waiting list for treatment or therapy.

[25] Compliance with treatment is an issue in any study as well as generally. Participants were rated for compliance of medication on a scale of 1 (none) to 8 (full). 77% of participants had a median score of 7 or 8, and there was no difference in compliance between the two groups.

On the Clinical Global Impression Improvement Scale (CGI-I) (Guy 1976) [26], both groups showed similar improvements by 28 weeks (table 20.3).

	SSRI ALONE	SSRI AND CBT
Very much, much improved	60.7	53.1
Minimally improved, no change, minimally worse	31.9	40.7
Much worse, very much worse	7.4	6.2

(After Goodyer et al 2007)

Table 20.3 - Percentage of participants at week 28 on CGI-I.

So, overall, CBT with SSRI was not significantly better than SSRI alone for major depression among adolescents.

APPENDIX 20A - CBT

CBT has a number of strengths and weaknesses (table 20.4).

STRENGTHS	WEAKNESSES
1. Concentrates on thinking. 2. Based on testable theory that maladapted thinking behind psychological problems and mental disorders. 3. Clear goals of reducing or removing symptoms. 4. Concentrates on the "here and now".	1. Neglects emotions with emphasis on thinking. 2. Neglects past history with focus on "here and now". 3. No attempt to find the underlying cause of problems. 4. Depends upon the client's ability to reflect, monitor and control their thoughts/thinking patterns.

Table 20.4 - Strengths and weaknesses of CBT.

Not all CBT is the same. Kanter et al (2005) found that when the therapist focused more on the therapeutic process rather than the relationship with the client, the CBT for depression was perceived as more effective by the

[26] The CGI-I scored from a combination of parents' reports and sufferer self reports.

clients. However, scores on the Beck Depression Inventory did not show improvements.

While Gassman and Grawe (2006) found that successful therapists generally concentrated on the client's strengths ("resource activation") most at the beginning of sessions and then their problems ("problem activation"). Based on analysis on 120 video-recorded therapy sessions, unsuccessful therapists were the opposite.

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SUFFERING THE TRAUMA OF A DISASTER AND THE EXPERIENCE OF BEING HELPED

EXAMPLE FROM SRI LANKA

Sri Lanka was badly hit by the Boxing Day (26th December) 2004 tsunami which killed an estimated 31 000 people (Bailey 2008). Survivors experienced the after-effects as complaints of physical symptoms without physical causes, like chest and abdominal pains, headaches, and fatigue. These types of physical symptoms are part of what are is categorised as post-traumatic stress disorder (PTSD) in the West.

The core symptoms of PTSD are re-experiencing the event (eg: flashbacks), emotional numbing, and hyperarousal (eg: easily startled) (APA 2000).

There was an influx of foreign relief workers and volunteers including mental health workers to help with the psychological problems. But some of the methods of the latter group were "not always culturally sensitive or locally appropriate" (Bailey 2008).

Researchers also came to study the effects of the tsunami with projects that had not received full ethical clearance as there was no central research ethics committee in Sri Lanka (Sumathipala and Siribaddana 2005). This interest was in contrast to the usual lesser interest in Third World countries.

Neuner and Elbert (2007) noted three reasons for the lack of research of and help for people living in traumatised regions of the world:

- "The Western world does not benefit from research in this area and in science it is considered exotic, an 'Orchideenfach' (orchids are beautiful but bear no fruit)".

- "Reviewers often argue that rigorous scientific standards cannot be met in developing countries and conflict regions. There are too few qualified researchers to collaborate with. There are no reliable sources to be used for proper sampling, no telephones available for random-digit dialling".

- Western intervention is seen as undesirable and intrusive. But "the consequences of not interfering in cultural norms would also include withholding knowledge about scientific methods of objective assessment and evaluation from these cultures".

In terms of the studies of PTSD, Neuner et al (2006) found 15% of children opportunity sampled had symptoms 3-4 weeks after the disaster. While 41% of adolescents and

20% of mothers showed symptoms 4 months after (Wickrama and Kaspar 2007). Hollifield et al (2008) investigated 20-21 months after the event in the badly hit southern area of Sri Lanka. Eighty-nine adults from 61 homes were randomly sampled in the Peraliya area. The rate of clinical PTSD was 21%. PTSD symptoms were correlated with thinking life was in danger during tsunami, and injury to and/or death of family member, but not of friends or property damage.

It was found that respondents coped using culturally relevant resources including family and friends (56% of respondents), own strength (70%), and Western-style hospitals (56%).

SOCIAL SUPPORT

"Humans experience traumatic events differently from all other animals because we ascribe meaning to events that befall us" (Charuvastra & Cloitre 2008 p303). Not all traumas are equal in terms of the risk of PTSD, and injuries caused by other people are the traumatic events most likely to result in PTSD. For example, in US studies, about half of rape victims develop PTSD compared to around 10% of those in natural disasters (Charuvastra & Cloitre 2008).

Charuvastra & Cloitre (2008) described the social ecology of PTSD (ie: the social context of the disorder) which includes social support.

Social support protects against PTSD developing, and the lack of social support increases the risk. For example, Koenen et al (2003) studied US soldiers returned from the Vietnam War. Individuals who reported greater community involvement had greater remission of PTSD symptoms, whereas those who experienced the community as negative towards them had more symptoms.

While Borja et al (2006) found that adult sexual assault victims with negative social interactions had a greater risk of PTSD, but those with positive interactions reported "post-traumatic growth" (eg: greater appreciation for life or spiritual development). So positive and negative social support and interactions (or perceptions of them) have different patterns of influence (Charuvastra & Cloitre 2008).

However, it is not quite as straightforward as that. The absence of social support or negative social interactions always leads to negative outcomes in terms of PTSD. But positive social support depends upon factors like who gives the support, whether the support offered is what is needed, and the nature of the trauma (eg: sexual assault with stigma and shame associated or a

shared, unambiguous event like a terrorist attack) (Charuvastra & Cloitre 2008).

It seems that as the individual recovers from a trauma, positive social support conveys the message that the individual is cared for and protected, whilst negative support reinforces the feelings of insecurity and fear. Avoiding behaviour is also affected by social support with negative support encouraging continued avoidance of associations with the trauma (Charuvastra & Cloitre 2008).

Studies have confirmed the importance of support in the form of the family/caregiver for the child victim. For example, among 72 child with acute burns, separation anxiety (distress over separation from parent(s)) was significantly associated with PTSD symptoms (Saxe et al 2005). But there is a problem if the parent is the source of the trauma (eg: as in abuse): "the parent is both agent of the trauma and an effective agent of comfort, creating a paradoxical or confusing state that further undermines the child's sense of safety" (Charuvastra & Cloitre 2008 p311).

Scott (1999) described how such confusion can occur with Dissociative Identity Disorder [27] among young women subjected to ritual abuse in the family and/or in a cult: ".. a two, or three-way split between life at school, and life at home or in an abusive cult context. At home, life might have been further split between time which involved keeping up appearances and 'abuse time'. In some families cult activities and beliefs were never discussed at home; even visible injuries would not be mentioned, and small children would be told they had dreamed or imagined any abuse they referred to" (pp448-449).

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[27] The key symptom of DID is "the presence of two or more distinct identities or personalities or personality states (each with its own relatively enduring pattern of perceiving, relating to and thinking about the environment and self)" (APA 1994 p487 quoted in Scott 1999 p436).

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22. SHYNESS: NORMAL OR ABNORMAL?

Chronic shyness is a fear of negative evaluation which causes emotional distress and inhibits an individual's life (Al Razek et al 2006). Buss (1985) distinguished between fearful shy individuals who are afraid of novelty, and self-conscious shy individuals who have an excessive focus on public aspects of the self.

Al Razak et al (2006) preferred to distinguish between a normal (physiological) shyness and the abnormal (pathological) type which overlaps with social phobia [28], and avoidant personality disorder (box 22.1).

Half of the population experience shyness, but only just over 10% are disable by it (Henderson 2004). While in a US study, 40% of individuals self reported as being chronically shy, 40% as previously but no longer, 15% as shy in some situations, and 5% never shy (Zimbardo et al 1974).

Avoidant personality disorder is a "pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation" (APA 1994). DSM-IV diagnoses it based on four or more symptoms from the following seven:

- Avoids interpersonal contact
- Unwilling to form relationships
- Restraint within intimate relationships
- Preoccupation with being criticised or rejected
- Inhibited in new interpersonal situations
- Views self as inferior to others
- Risk adverse

Box 22.1 - Criteria of avoidant personality disorder.

Al Razak et al (2006) investigated shyness among 76 Egyptian medical students and 76 Saudi Arabian college students all aged between 18-23 years. The volunteers completed the Shyness Scale, Fear of Negative Evaluation Scale, Social Phobia Scale, and the Diagnostic Checklist of Personality Disorders (all Arabic versions).

Fifteen percent of the sample were classed as showing pathological shyness with it more common among women. Shyness Scale scores were significantly positively correlated with Social Phobia Scale and Fear of Negative Evaluation Scale scores.

[28] Social phobia first appeared in DSM-III in 1980, and defined as "a marked and persistent fear of one or more social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The individual fears that he or she will act in a way (or show anxiety symptoms) that will be humiliating or embarrassing" (APA 1994 p416 quoted in Scott 2006 p135).

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The line between normal shyness and pathological shyness or diagnosis with social phobia and avoidant personality disorder has a lot to say about how behaviour in a society is defined as normal or abnormal.

Scott (2006) argued that there was a medicalisation of shyness into social phobia, and this allowed for the use of medications, like SSRI anti-depressants, with it. She believed that shyness is seen as "a failure to achieve certain cultural values, such as assertiveness, self-expression and loquacious vocality". For her, shyness lies on the "contested boundaries" between physical health, mental illness, and social deviance:

> On the one hand, this is a relatively normal experience: many of us can identify with episodic feelings of shyness that arise in certain types of situations. On the other hand, some people identify so strongly with the "shy" label that they feel constantly anxious, lonely and frustrated, and understand shyness to be a chronic and debilitating condition that interferes with their everyday lives" (Scott 2006 p133).

The increasing number of self-help books, websites, and therapies present shyness as "a new social problem, of apparently epidemic proportion". The reticence associated with shyness is seen as a barrier to a "successful life", which is, thus, a "neglect of social responsibilities". "As a reflection of changing social values, therefore, the medicalisation of shyness suggests that bashful modesty and reserve are no longer so acceptable and that to succeed we must be vocal, assertive and capable of gregariously participating in social life" (Scott 2006 p134).

The distinction between normal and abnormal can be viewed in different ways:

1. Deviation from the social or statistical norm is abnormal.

Either social or statistical criteria are seen as the norm, and the absence of these is abnormal. For example, a certain amount of social interaction without fear is normal, and individuals who cannot do this are abnormal. Statistical criteria uses the majority, for example, as the norm.

Problems with deviation from social norm:

i) Depends on what society decides is normal, not necessarily what is a problem for an individual. For

example, in Nazi Germany norms were established by the government that encouraged hatred and mistreatment of Jews, Romany people, and those with disabilities. In particular, these groups had to wear distinctive clothing and the norm for Germans was to spit at them (Brewer 2001).

ii) It can be a means of social control for those individuals who do not conform. Conversely, there may be individuals who conform but are suffering from a problem (eg: "conforming neurotics"). Part of the problem is a fear of rejection, and so they adapt their behaviour to fit in perfectly (Brewer 2001).

iii) Definitions will vary between cultures, societies, and historical periods, so no universal criteria. For example, in Victorian England (nineteenth century), women wore between six to eight undergarments to make sure that every part of their body was covered, except the face. In particular, the female leg was seen as highly arousing for men, such that even table legs were covered, and the word "limb" was used instead of "leg" in polite company (Brewer 2001).

Problems with deviation from statistical norm:

i) How to establish statistical norm - for example, by absolute majority or standard deviations from norm using the normal distribution (box 22.2).

ii) A behaviour may be statistically normal, but not desirable. For example, a majority of people will suffer from some form of depression at some stage in their lives. It is usually felt that depression is not a desirable or normal state of affairs. Controversially, it has been suggested that over half of the USA population have experienced abuse of some kind as a child. Again this may be statistically normal, but not desirable.

iii) No universal criteria as definitions will vary between cultures, societies, and historical periods.

2. Presence of symptoms

The diagnosis of mental illness is based upon the presence of certain symptoms seen as maladaptive. It does not matter how may people have the symptoms, their presence is enough for a diagnosis of abnormal (mentally disordered). For example, refusal to meet people through fear of embarrassment while desperate to interact socially is a symptom.

By far the most common statistical technique is the normal distribution. This is a statistical concept which is able to establish the scores of a majority of people on a particular test. It is most often used for intelligence. Any IQ test will have a mean score (usually standardised to 100), and a standard deviation (which shows how the scores cluster around the mean).

It has been statistically established that certain percentages of the population can be found at certain scores (standard deviations). Statistically 95% of the population will score between -2 and +2 standard deviations of the mean. This is classed as the normal range. Using an IQ test with a standard deviation of 15, this would make the normal range vary from 70 (-2 standard deviations) to 130 (+2). Therefore anybody with an IQ outside that range is classed as abnormal.

There are problems, though. Firstly, IQ tests vary on their standard deviations. For example, the Stanford-Binet test has a standard deviation of 16, while for the Wechsler Adult Intelligence Scale (WAIS), it is 15. Another test may have a standard deviation of 10. In each case, there will be a different range that is classed as the normal range.

TEST	A	B	C
MEAN	100	100	100
STANDARD DEVIATION	20	15	10
-2 TO +2 RANGE	60-140	70-130	80-120

Secondly, the decision to establish the normal range as -2 to +2 standard deviations is a subjective one. It could quite easily be -1 to +1 standard deviations (which covers approximately 68% of the population), or -3 to +3 standard deviations (98% of population approximately) (Brewer 2001).

Box 22.2 - Normal distribution of scores and establishing normality.

Problems with presence or not of symptoms:

i) Who decides that certain symptoms are abnormal? The answer is the medical profession, and especially psychiatrists, who view mental illness as the same as physical illness in the body. This is the medicalisation of behaviour.

ii) Do the presence of certain symptoms mean an underlying mental disorder exists? There is much debate about the reliability and validity of classification systems, like DSM-IV, that use symptoms to diagnose mental disorders.

iii) Gives power to the groups in society who do the diagnosing. This also allows for what is seen as abnormal

to expand and what is normal to be reduced. For example, in figure 22.1 , using the high and low emotions, new categories of mental disorder have limited the room for normal behaviour - major depressive disorder, mild depressive disorder, and minor depressive disorder.

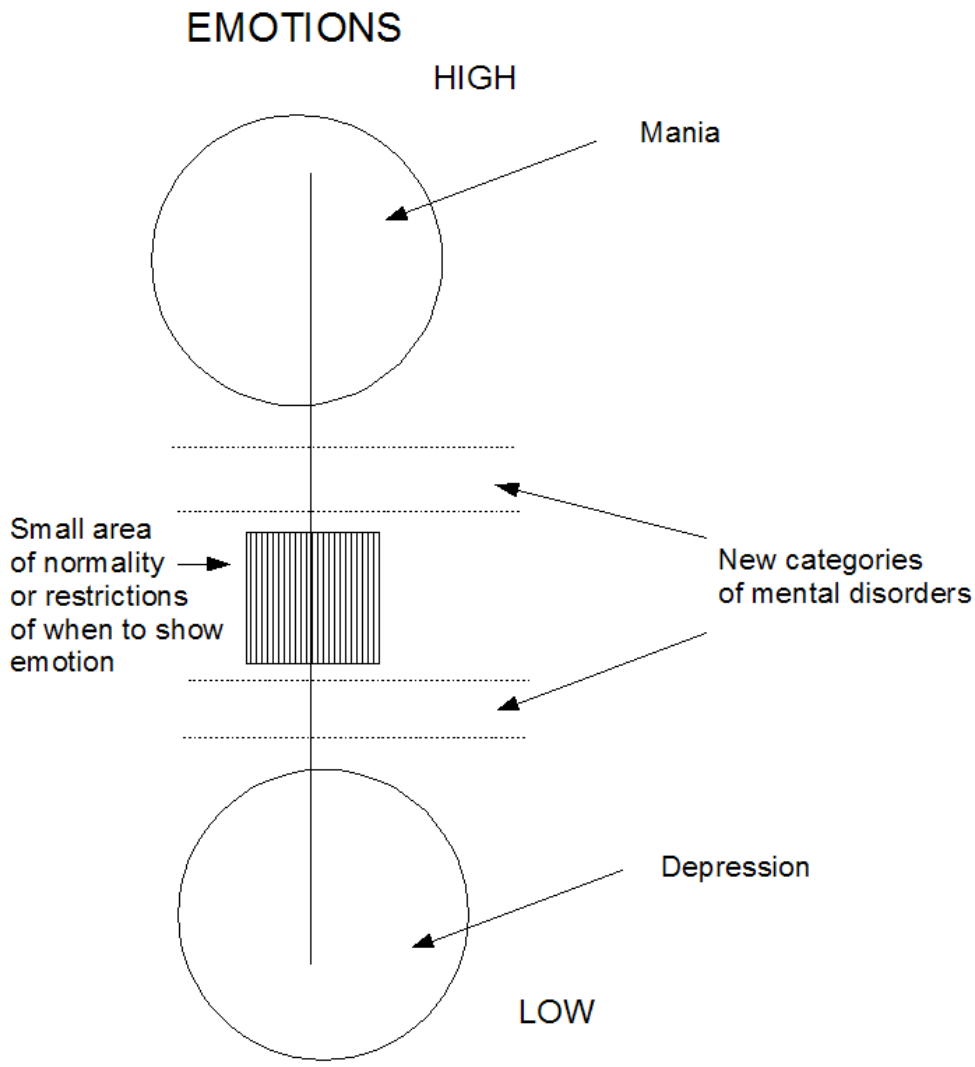

Figure 22.2 - How expanding categories of mental disorders for high and low emotions reduces the room for normal behaviour.

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23. STRESSFUL LIFE EVENTS AND DEPRESSION

The same negative life event to two people can lead to one of them becoming anxious and depressed, and the other coping with it without psychological problems. Work comparing identical and non-identical twins has suggested that part of the difference is due to genetic variations. The difference in "emotional resilience" is affected by a gene that is involved with levels of the neurotransmitter, serotonin. The "anxious version" of the gene leads to depression more often, but only after very difficult life experiences (Canli 2008).

Lesch et al (1996) found that the gene (SLC6A4 on chromosome 17) related to serotonin and its transportation [29] came in a long and a short version (5-HTTLPR). The long version leads to more serotonin transport (ie: more serotonin). Individuals who had higher anxiety scores were more likely to have the short version of the gene (ie: producing less serotonin).

Building on this observation, Hariri et al (2002) showed that the short version of the gene produces greater excitement in the amygdala (described as the "emotional part" of the brain). In a functioning magnetic resonance imaging (fMRI) scanner, individuals with the short version had a greater response in the amygdala to pictures of emotional faces (eg: fear, anger) than individuals with the long version.

In terms of the interactions of this gene with life events, Caspi et al (2003) compared the gene variants in 847 New Zealanders and their stressful life events between 21 and 26 years old. The short version (or more correctly, at least one short version of two possible alleles; ie: one from biological mother and one from biological father) was associated with depression after negative life experiences - eg: four or more traumatic events - 33% of short version group depressed versus 17% of those with two long versions (figure 23.1).

[29] Serotonin transporters capture serotonin after the synapse and return it to the pre-synaptic neuron. This is known as reuptake.

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BIOLOGICAL MOTHER	BIOLOGICAL FATHER		STRESSFUL LIFE EVENTS		DEPRESSION
short	short)	→	NO	→	NO
short	long)				
long	short)	→	YES	→	**YES**
long	long)	→	NO	→	NO
	)	→	YES	→	**NO**

Figure 23.1 - Version of 5-HTTLPR gene and depression after stressful life events.

The mechanism by which stressful life events lead to depression seems to be rumination (ie: preoccupation with thoughts about the negative events). The short version of the gene and life stresses were associated with a greater tendency to ruminate in response to moody facial expressions as the fMRI scans showed a more active brain. Individuals with two long versions of the gene were less likely to dwell on things (Canli and Lesch 2007).

The differences in the serotonin transporter gene only accounts for part of the variation in response to stressful life events, and other factors are involved. These include fifteen other genes (Canli 2008). Another factor is coping strategy, for example.

Tait et al (2004) showed that "sealing-over" (avoidant) coping strategy as opposed to integration was associated with negative childhood experiences and insecure adult attachment, and with low resilience to life events. Coping strategy was measured by the 39-item Recovery Style Questionnaire (RSQ) (Drayton et al 1998) among fifty individuals with schizophrenia.

Integration is where the individual thinks about their negative experiences and puts them into context, while sealing-over involves not thinking about such experiences during recovery (McGlashan et al 1976). Practically, individuals using this coping strategy when recovering from psychosis are less engaged with mental health services.

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24. NAMES AND LABELS AND SCHIZOPHRENIA

The term "schizophrenia" was first coined in 1911 by Eugen Bleuler [30]. So nearly one hundred years old, and the feeling, for many people, is that it is an unhelpful term, particularly because of the stigma attached to it [31]. There is a desire to change the term to emphasise the variety of experiences of sufferers. Suggestions include stress-sensitivity psychosis, traumatic psychosis, and anxiety psychosis. In Japan, the term meaning "integration disorder" was introduced (Kingdon 2007) [32].

"Renaming schizophrenia is highly controversial. It is difficult for psychiatrists and researchers to acknowledge that the concept they have been using for almost one hundred years makes no sense" (Kingdon 2007).

The label of schizophrenia is meant to be a technical term used by psychiatrists which describes clearly a specific illness in the same way as any other medical term. But this term is so often used in society, inappropriately in many cases, and has negative associations with it. One woman diagnosed with schizophrenia interviewed by Dinos et al (2004) said: "Schizophrenia is the worst diagnosis because I've heard it in the newspaper and on TV, that they are really mad schizophrenic people, they are very dangerous to society, they've got no control" (p177). Forty-six individuals were interviewed and many reported concerns about telling others that they had a diagnosis of schizophrenia - eg: "..if anybody at work or my professional body knew that I'd got schizo-anything I wouldn't be allowed to practise" (Dinos et al 2004 p178).

Names and labels are not neutral descriptions of terms, whatever the intention of those who devise them, because language has social meaning (box 24.1). More than that, argued Linnett (2004): "The problem of language of mental health is complex. It goes far beyond the use of derogatory expressions or whether we should call people 'service users' or 'clients'. It highlights the confusion and weak foundations of psychiatry.." (p12). Many labels are value judgments rather than objective categories:

> Lay people and psychiatrists alike tend to call people mentally healthy when they like their behaviour and mentally ill when they dislike their behaviour.

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[30] Initially, "dementia praecox" (early-onset dementia) was used.

[31] See Campaign for Abolition of the Schizophrenia Label (CASL) (http://www.asylumonline.net/).

[32] In Holland, the term "perception dysfunctional syndrome" (PDS) has been adopted by the user group Anoiksis (http://www.anoiksis.nl/).

> Rebellious teenagers, unhappy housewives, dissatisfied workers, or lonely old people, for example, are often diagnosed as mentally ill, which is less a medical, scientific description than it is a judgment that the person so labelled has, in some way, behaved improperly (Chamberlain 1988).

- Deficiency metaphor - eg: "screw loose".
- Replacing of sanity by something else - eg: "bats in the belfry".
- Transition from sanity - eg: "cracked".
- Similar to animal behaviour - eg: "cuckoo".
- Based on behaviour - eg: "berserk" (from Beserker, Norse warrior, who fought with frenzied rage).
- Where reason lives - eg: "off your nut" (nut = head).
- Transport metaphor - eg: "off the rails".
- Places - eg: "south of the border".

Box 24.1 - Different forms of language used to describe mental illness in everyday life (Leff 2000).

Chadwick (2003) observed that DSM-IV "contains very few diagnostic labels that put psychiatrists, scientists and psychoanalysts in a bad light". So the Bloomsbury Artistic Psychology Association proposed, tongue-in-cheek, their own diagnostic categories for DSM-V including "pathologically middle-of-the-road personality disorder" (MORPID), "totally colourless personality syndrome" (TOCLOPS), and "fixation on middle class normative values".

In a similar vein, Tarragon et al (2006) proposed the category of "service user consultant disorders" to describe the negative attitude of psychiatrists towards service users as consultants or "experts by experience". Symptoms included "an intense and irrational fear of service user consultants (SUCs)", "irritability in presence of SUCs", and "poverty of speech (tendency to communicate little in the presence of SUCs)".

Even if these ideas were entirely serious, they probably would not catch on because those in power are able to apply labels that stick not the powerless.

> For example, if some students defined price-fixing and profiteering among corporate executives as deviance and a problem in need of remedy, they ordinarily would not have the power to implement their definitions of the situation (Conrad 1981 pp111-2).

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25. MEASURING THE AMOUNT OF SUBSTANCE USE ACCURATELY

Obtaining accurate measures of substance use is difficult because, in the case of illegal drugs, users keep themselves hidden, while generally, it is better for users to downplay the amount. So finding individuals to ask is difficult, and those who are found may not be honest. This poses a problem for researchers seeking to gain accurate (true) measures of behaviour. Consequently, alternative methods to simple self-reports are required. This article describes two such techniques.

1. Prevalence of crack cocaine use in London.

Government figures suggested a prevalence of 0.3% in London from self-reports (Aust and Condon 2003), but this is seen by researchers as an underestimate.

Hope et al (2005) used the capture-recapture method and a ratio-estimation method to calculate a more "accurate" figure of 1.3%. These techniques estimate the size of a group based on statistical assumptions.

Firstly, the information that is known is collected - crack cocaine users in the twelve London boroughs in 2000-2001 - from "arrest referrals" for substance problems at police stations, and overdoses at Accident and Emergency Hospitals. Then calculations are made of the rate of observed/known to unobserved/unknown based on knowledge about injecting drug users generally and the number of them using crack cocaine [1]. For example, 4117 cases were observed and 16 855 were calculated as unobserved in the twelve London boroughs, which is an estimate of 47 000 users for the whole of London.

2. Amount of substance use in the USA.

It is quite probable that individuals will under-report both illegal and legal substance use when asked. A more accurate measure is toxicological screening. Rockett et al (2006) were able to compare both types of measures for individuals in seven Tennessee hospital emergency departments (not in police custody).

1502 adults gave self reports of their substance use, and saliva and urine samples. It was not a representative sample of the whole population because

[1] Total number of crack cocaine users = Proportion of crack cocaine users that are injecting drug users divided by Proportion of injecting drug users that are crack cocaine users x Size of injecting drug-using population (Hope et al 2005 p1704).

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only those at the hospitals were studied. Overall, there was under-reporting for all substances compared to toxicological screening with the least being for alcohol and the most being for marijuana, cocaine, and stimulants (table 25.1). The under-reporting was most evident among older adults (appendix 25A).

	MALE		FEMALE	
	SELF-REPORT	SCREENING	SELF-REPORT	SCREENING
ANY SUBSTANCE	60.9	69.1	43.9	55.7
ALCOHOL	45.9	46.6	25.1	25.5
MARIJUANA	10.6	21.4	5.9	13.0
COCAINE	1.1	6.5	0.6	3.5
STIMULANTS	1.5	3.5	2.1	9.9

(After Rockett et al 2006)

Table 25.1 - Percentage of use for selected substances based on self-reports and toxicological screening.

Both these methods have strength and weaknesses compared to the traditional use of self-reported surveys (table 25.2).

CAPTURE-RECAPTURE METHOD	TOXICOLOGICAL SCREENING
Strengths 1. A better estimate of users among hidden population. 2. Shows under-estimation of official statistics. Weaknesses 1. Is only an estimate. There is no way to confirm accuracy. It could be an over-estimation or an under-estimation. 2. Depends upon the assumptions of the statistical model used.	Strengths 1. Toxicological screening generally accurate. 2. Individuals may not remember when self-report and screening overcomes this problem. Weaknesses 1. Toxicological screening depends on factors like time since use and substance, so not perfect. Possibility of false positivity (ie: reports substance present when not). 2. Depends upon consent of participants. Individuals who use substances could have refused.

Table 25.2 - Strengths and weaknesses of alternative methods to self-reports.

APPENDIX 25A - STUDYING OLDER ADULTS AND MEDICATION

Rockett et al (2006) found an under-reporting of drug use by older adults compared to the toxicological screening. Typically studies of the use of drugs by different age groups is cross-sectional. Groups of different ages are sampled at one point in time and compared. This type of study does not show the pattern of use by individuals over time which longitudinal studies can show. This is also the case with Rockett et al.

Stowell et al (2008) concentrated on benzodiazepine (tranquilliser) use over a twelve-year period among 1681 individuals aged 65 years and above in Pennsylvania, USA [2]. Use of the drug was based upon researchers viewing participants' prescriptions at each data collection point (biennial).

Initially, 5.5% of men and 9.8% of women were taking the medication. Sustained use was more common among women, users of multiple other medications, and smokers. The study did not measure over-the-counter medication use.

The number of participants was reduced to 40% of the original sample by the end of the study, mostly by death, but some individuals chose not to continue in the study. This highlights a general problem in both the recruitment and retention of older adults in research.

Mody et al (2008) listed some of the factors involved:

- Health and mobility problems that limit availability and willingness to participate;
- Motivation to participate;
- General mistrust of researchers and authorities (eg: fear of scams if door-to-door recruiting);
- Reactions of "gatekeepers" (eg: family members or service providers restricting participation);
- Fears of privacy not being protected.

Some of these issues are common to all participants (eg: motivation), others may be specific to older adults.

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[2] 665 participants remained by the end of the study.

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26. STRESS-RESPONSE SYSTEM AND CHILDHOOD ANTI-SOCIAL BEHAVIOUR

The starting point for van Goozen et al (2008) was that anti-social individuals are less sensitive to stress as shown by less fear to engage in risky and dangerous behaviour, and of the negative consequences from it.

Zuckerman (1979) talked of sensation-seeking behaviour among individuals who are easily bored and less discouraged by potential danger. Such individuals have a higher threshold for stress (eg: low resting heart rate).

For example, Raine et al (1997) measured resting heart rate at three years old among 1700 children in Mauritius, and then their level of aggression eight years later. Low heart rate at three years predicted aggressive behaviour at eleven years old.

van Goozen et al (1998) compared children diagnosed with conduct disorders and those not on a fixed video game. Children individually played against an "opponent" (reactions fixed by experimenters) which produced competition, who antagonised the child, and frustrated them from winning. Physiological measures that were taken showed lower heart rate, skin-conductance and cortisol reactivity to the stress by children with conduct disorders (physiological hyporeactivity), but they showed more aggressive behavioural responses (appendix 26A).

The physiological hyporeactivity could be a product of a stressful early life. "A down-regulation of the stress-response system in the face of chronic stress in early life would be an adaptive mechanism, avoiding chronic arousal and excessive energy expenditure that could ultimately result in serious pathophysiological consequences" (van Goozen et al 2008 p227).

Ortiz and Raine's (2004) meta-analysis of forty studies (box 26.1) confirmed a significant relationship between low resting heart rate and child anti-social behaviour (overall effect: d = -0.44). The effect was negative because the correlation is a negative relationship (ie: lower resting heart rate and higher anti-social behaviour and vice versa). The strength of the relationship was not influenced by measuring techniques for heart rate or rating of aggression (eg: self or teacher). A lower heart rate was also found among anti-social children during stress.

A number of other points were made by Ortiz and Raine (2004):

- No gender differences in relationship between resting heart rate and anti-social behaviour;

- The relationship was found in studies in seven different countries: USA, Canada, England, Germany, New Zealand, Mauritius, and Siberia (Russia);

- The relationship has been found in other mammals including macaques, baboons, and rabbits;

- Some of the studies are prospective (ie: heart rate taken at age before anti-social behaviour develops), and this rules out the possibility that anti-social behaviour caused the low heart rate;

- On the other hand, high heart rate protects against adult anti-social behaviour;

- There is evidence of offspring of criminal parents having low resting heart rate.

438 articles found with key word search using terms: heart rate, pulse, cardiovascular, autonomic nervous system, antisocial, aggression, externalizing, violence, delinquency, conduct disorder, and oppositional defiant disorder.

This was reduced to 40 by inclusion and exclusion criteria. "Inclusion criteria used were as follows: (1) sample consists of children or teenagers under age 19 years; (2) minimum of 10 subjects in a sample; (3) subjects must have been assessed systematically on some form of antisocial behaviour; (4) heart rate was assessed under a rest condition (studies that also assessed heart rate during a stressor were flagged for further evaluation; see below); and (5) use of either a correlational or between-group design.
Exclusion criteria were as follows: (1) insufficient information about the heart rate–antisocial relationship to allow effect sizes to be computed (unless the information could be obtained directly from the author); (2) results reported from a sample already included in the meta-analysis (in case of multiple papers reporting on the same population, the study with the largest sample was chosen for inclusion); and (3) case reports and review articles" (Ortiz and Raine 2004 p155).

The total sample size was 5868.

Box 26.1 - Details of inclusion and exclusion criteria of meta-analysis.

APPENDIX 26A - VAN GOOZEN ET AL (1998)

This Dutch study compared 21 boys (8-11 years old) diagnosed with oppositional-defiant disorder (ODD) (DSM-IV criteria) and thirty-one similar aged matched IQ controls from the Utrecht area.

The experiment lasted 2½ hours [3] divided into three phases:

i) Baseline measures of heart rate and initial saliva sample while filling in questionnaires and playing non-stressful video games (45 minutes).

ii) The experimental manipulation of stress (75 minutes). Frustration was induced by the pressure to solve an unsolvable task in a limited time while in competition with another player. The other player was a videotape of a boy of a similar age who criticised the player's performance (provocation). Aggression was measured as the frequency and intensity of white noise given to the "competitor". A saliva sample was taken during this session, and heart rate measured throughout.

iii) Debriefing (30 minutes). The child was told how well he had performed and given a prize. A saliva sample was again taken as well as the continued heart rate measure. There are no details of whether the boys were fully informed here of the deception (ie: no real competitor).

The cortisol level (which is a measure of stress; increased stress = increased cortisol) in the saliva was lower throughout the experiment in the ODD boys. These boys had lower resting heart rates but higher heart rate during the stress (suggesting more anger).

There are ethical concerns about this research because the children were being deceived and deliberately frustrated and provoked to aggression. van Goozen et al reported that the study was approved by the Medical Ethics Committee of Utrecht University Hospital, and parents were asked to give written informed consent for their child's participation.

In Britain, the BPS (2006) code of conduct says: "Consider all research from the standpoint of research participants, for the purpose of eliminating potential risks to psychological well-being, physical health, personal values, or dignity" (p18).

This type of research is common, but are the children fully protected from potential risks? Furthermore, how effective is the debriefing in removing after distress?

[3] Always 9-11.30 am to control for natural circadian changes in physiology over the day, like cortisol levels.

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27. ANIMAL MODELS FOR MENTAL DISORDERS

Studying non-human animals (animal models) to understand human behaviour is common throughout psychology. The physiology of mammals, for example, is similar to humans, and it is possible to treat them in ways not acceptable with human participants (eg: brain damage, severe sleep deprivation). But they are not humans, and this seems most important in the case of mental disorders. These disorders appear to be relatively unique to humans, or certainly the ability to describe the experience of them, so what use are animal models here? This article describes two recent examples of animal research to offer as an answer to this question.

IMMUNE SYSTEM AND COGNITIVE DYSFUNCTION IN MICE

Kipnis et al (2004) were interested in the role of immune deficiency on cognitive dysfunction. In other words, if the immune system is not functioning properly this can lead to cognitive problems.

Specially inbred and knockout mice were used that had immune deficiency, in particular, a shortage of T cells (which fight off "invaders" in the bloodstream). Cognitive abilities were tested with the Morris Water Maze (MWM) test. The animal is placed in a tank of water and has to find a hidden platform. Initially, it is trial and error, but on subsequent trials, how quickly they find the platform is a measure of spatial learning/memory.

The mice with T cell shortages took longer to find the platform on subsequent trials than control animals (eg: mean of 80 vs 20 seconds on day 4). This showed that normal immune function is involved in normal cognitive processing.

The mice were then given a vaccination that stimulated T cell development, and their performance on the MWM test improved (ie: no cognitive dysfunction). The researchers believed that this vaccination may aid individuals with AIDS who show cognitive deterioration.

The vaccination was also able to combat amphetamine-induced psychosis in the mice (similar to schizophrenia in humans, it was argued).

This study has a number of advantages over using human participants:

i) Able to study specific immune deficiency with specially bred animals;

ii) Able to control the environment and testing, and reduce outside influence/extraneous variables;

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iii) Injections to induce psychosis-like symptoms and vaccinations possible to see the effects;

iv) Less concern about side effects and risk of injections than with humans;

v) The basic brain physiology of mice is the same as humans.

REWARD LOSS AND DEPRESSION IN RATS

Burman et al (2008) induced negative affect in rats to simulate depression.

Twenty-four rats were kept in two different cages for the period of the experiment. One caged was the enriched environment throughout (E), while the other cage had objects removed to induce negative emotions (U - unenriched).

The experimental task involved running along a two-metre runway to gain food. All the animals were trained for twelve days to receive twelve food pellets. Then on the thirteenth day, the food reward was unexpectedly reduced to one pellet (this was the reward loss). The one pellet reward continued for five more days. The focus was upon how the rats would react to the reward loss.

All animals ran significantly slower on day 13 on the trials after the reward loss. The E rats subsequently increased their speed each day, but the U rats took longer to return to a faster speed, particularly on days 15 and 16 (table 27.1). Thus they were more sensitive to the reward loss.

DAY	E RATS	U RATS
12 (reward of 12 food pellets)	5	5
13 (reward of 1 pellet introduced)	10	10
14	16	14
15	12	20
16	10	20
17	10	10

Table 27.1 - Approximate mean speed (seconds) of rats along two-metre runway to food.

Table 27.2 lists the main arguments against animal models of mental disorders.

1. However close in terms of physiology, they are not humans.

2. Only able to induce schizophrenia-like psychosis, for example or simulate depression. Human depression is more complex than just a loss of desired objects.

3. Human mental disorders are about the experience for sufferers. Animal models cannot show this because language is the means of communicating the experience.

4. Tests used are of quite limited abilities rather than the full complexity of human cognition and behaviour.

5. Ethics of studying and treating animals in this way.

Table 27.2 - Arguments against animal models of mental disorders.

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28. AN UNORTHODOX TREATMENT FOR DEPRESSION

The use of drugs to treat depression is common in the form of anti-depressants (appendix 28A). But there are reports of benefits from other drugs, like ketamine.

Ketamine was developed as an anaesthetic, but appears to help depressives in hours rather than weeks which is usual with anti-depressants (Szalavitz 2007) (appendix 28B). It was first noted in rats that one dose stopped the development of behavioural despair (equivalent to human depression) in tests for over a week (Yilmaz et al 2002).

Berman et al (2000) were the first to try with humans using a single infusion of ketamine with seven treatment-resistant depressives, where two or more medications had failed. Within three days all the individuals showed improvements which lasted about a week.

Zarate et al (2006) (box 28.1) found similar improvements within hours among twelve of eighteen extreme treatment-resistant depressed individuals which lasted about a week again for some individuals after a single infusion. Previously ineffective anti-depressants were found to help after this trial (Szalavitz 2007).

If ketamine does work for depression, then the question is how is it working. Possibilities include the blocking of NDMA (N-methyl-D-aspartate) receptors in the brain preventing glutamate crossing the synapse [4] or that it activates another glutamate receptor called AMPA. Or that it increases the level of brain-derived neurotrophic factor (BDNF) (Szalavitz 2007).

- Individuals with major depressive disorder recruited in Washington DC area.
- Treatment-resistant = failed at least two anti-depressant trials.
- Given one dose of placebo then one week later one dose of ketamine (or vice versa).
- Outcome measures based on scores on Hamilton Depression Rating Scale (HDRS) (Hamilton 1960) - response = 50% decline of baseline score, remission = score of <7.
- Within 110 minutes significant difference to placebo.
- One day after: 12 responded/5 remission.
- One week after: 6 responded still.
- Two weeks after: 2 responded still
- There were immediate side effects including perceptual disturbance, confusion, blood pressure increase, and dizziness.

Box 28.1 - Details of Zarate et al (2006).

[4] Ketamine hydrochloride is a NDMA antagonist (ie: stops neurotransmitter), while agonist drugs mimic neurotransmitters.

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The lure of fast-acting anti-depressants is attractive, and other substances have been tried with success; eg: scopolamine hydrobromide (developed to treat motion sickness).

Furey and Drevets (2006) performed two pilot studies with individuals having major depression and depression as part of bipolar disorder. In study 1, eight individuals were given three doses of scopolamine and one dose of placebo with 3-5 days apart. The mean score on the Montgomery-Asberg Depression Rating Scale (MADRS) (Montgomery and Asberg 1979) decreased from 29.0 (baseline) to 17.6 after the fourth dose.

Study 2 involved eighteen individuals and seven sessions (3 placebo/4 drug). Full remission (50% of greater decline in MADRS score from baseline) after the seventh dose was shown by eleven participants and other seven showed partial remission (25-50% decline in MADRS score). Side effects of drowsiness, dry mouth, blurred vision, and lightheadedness were reported in both studies.

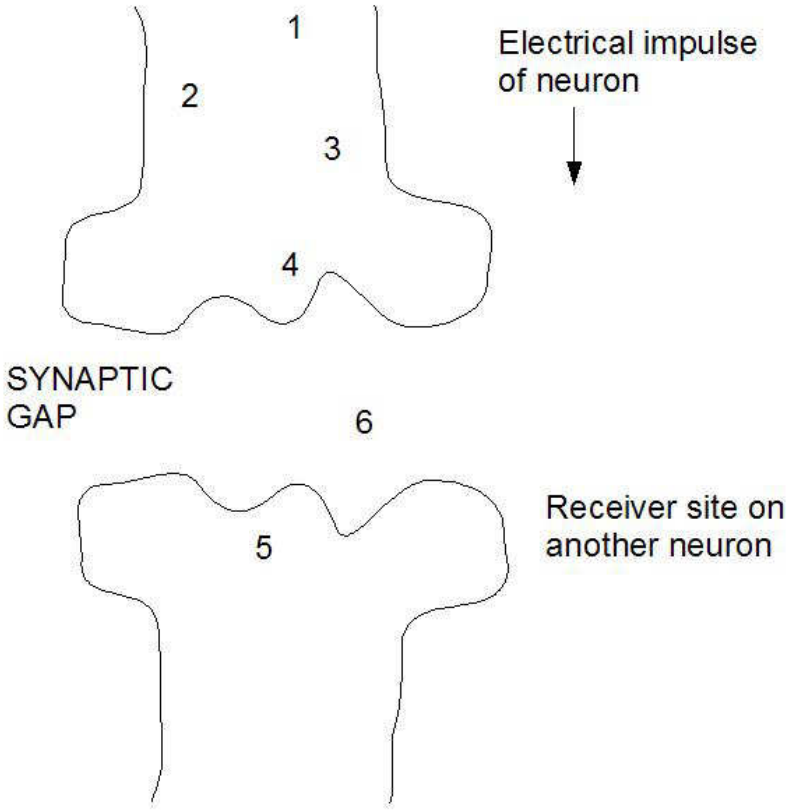
APPENDIX 28A - SYNAPSES AND ANTI-DEPRESSANTS

Drugs work on neurotransmitters at the level of the synapse. In other words, biochemical processes. Figure 28.1 shows the different points in the synapse where drugs can target for action.

APPENDIX 28B - SLOW ACTION OF ANTI-DEPRESSANTS

Anti-depressants usually take weeks of daily doses to have an effect on mood. Why does this happen? It does not seem to be due to neuropharmacology (ie: action of the drug on the brain). Harmer et al (2009) argued that the delay is a cognitive process (ie: the way the individual sees the world). The traditional view is that the pharmacology of anti-depressants improves mood which leads to cognitive changes (eg: interpreting events more positively). Harmer et al (2009) proposed that anti-depressants lead to cognitive changes first, then the improvement in mood. This way around accounts for the delay in effect of the drugs.

This is supported by, for example, an experiment where selective serotonin reuptake inhibitor anti-depressants given to healthy volunteers produced positive biases in cognitive processing (eg: tendency to interpret ambiguous facial expressions as positive), but the participants reported no change in mood (Harmer et al



(1) Synthesis/production of neurotransmitter in cell.
(2) Storage and transportation of neurotransmitter to synaptic gap.
(3) Incidental destruction of leaking neurotransmitters. MAO inhibitor anti-depressants stop destruction and thereby increase amount.
(4) Release of neurotransmitter across synaptic gap.
(5) Activation of post-synaptic receptors on other neuron. Ketamine blocks this process for glutamate, and thereby reduces activity.
(6) Deactivation and reuptake of neurotransmitters by firing neuron. Tricyclic, and selective serotonin reuptake inhibitor anti-depressants block different neurotransmitters here, and thereby increase amount.

Figure 28.1 - Synapse and points that drugs target.

2004). So, "rather than acting as direct 'mood enhancers', anti-depressants may re-tune how we process personal and socially relevant affective information" (Harmer et al 2009 p107).

If Harmer et al (2009) are correct then anti-depressants work in a different order to ketamine which produces an immediate benefit. It may be that ketamine changes the mood first, and this explains the short-term improvements (figure 28.2).

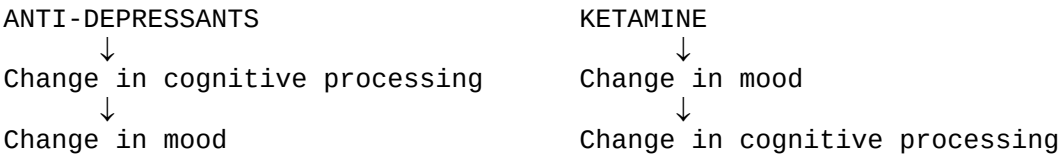


Figure 28.2 - Anti-depressants versus ketamine.

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29. WORRY GOOD, ANXIETY BAD?

The good news, said Curtis (2001) is that "all humans worry at some time or other, while 38% of Americans say they worry every day (Leahy 2005). What differs is its pervasiveness in terms of both frequency and intensity" (p29).

Borkovec (1994) defined worry as: "A chain of thought and images, negatively affect-laden and relatively uncontrollable; it represents an attempt to engage in mental problem-solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes" (quoted in Curtis 2001 p29). So worry appears to be focused on finding a solution to a problem.

But excessive worry is not good. "Chronic worriers operate under the misperception that their overthinking and attempts at controlling every situation allow them to problem-solve and plan for the future. Instead their thought pattern hinders cognitive processing and also causes overstimulation of emotion- and fear-processing areas of the brain" (Stern 2009 pp41-42).

In terms of anxiety, DSM-IV-TR (APA 2000) has a category called "Generalised Anxiety Disorder" (GAD) whose main symptom is chronic worry on most days along with three of the following in the past six months:

- Restlessness or feeling keyed up or on edge;
- Being easily fatigued;
- Difficulty concentrating or mind going blank
- Irritability;
- Muscle tension;
- Sleep disturbance.

Differences in brain activity have been shown in those who worry and are anxious. For example, Hofmann et al (2005) measured electrical activity in the brain in undergraduates told to give a public speech. The EEG showed more activity in the left prefrontal cortex before the event in worriers.

In an experiment using functional magnetic resonance imaging, individuals with GAD showed more amygdala activity leading up to seeing negative or neutral photographs than controls (Nitschke et al 2009). There was no difference between the groups at the sight of the pictures. The anxiety about the photograph had as much of an effect as the photograph itself for GAD sufferers.

Borkovec and Hu (1990) showed other physiological differences in worriers. There is an emotion-damping in

worriers which means their heart rate does not vary much between worrying, relaxing, or remaining neutral. The worry has dulled the emotional reaction. This means that such individuals are vulnerable to the physiological effects of stress as their body also takes longer to return to a resting rate after the event (Stern 2009).

Conceptualising worry and anxiety can be done in a number of ways.

i) A continuum from the good end of worry to aid focusing, planning, and problem-solving to the bad end of anxiety that has no positive benefits (figure 29.1 top). Somewhere there is a cut-off point between the two. So worry and anxiety are intrinsically the same. But this idea does not distinguish the differences between worry and severe anxiety.

ii) Worry and anxiety are separate experiences, which may overlap (figure 29.1 bottom). Within the worry circle, such behaviour is positive, while anxiety is always negative. An individual can move between the circles depending upon events in their lives, or some individuals may be more prone to one circle than another.

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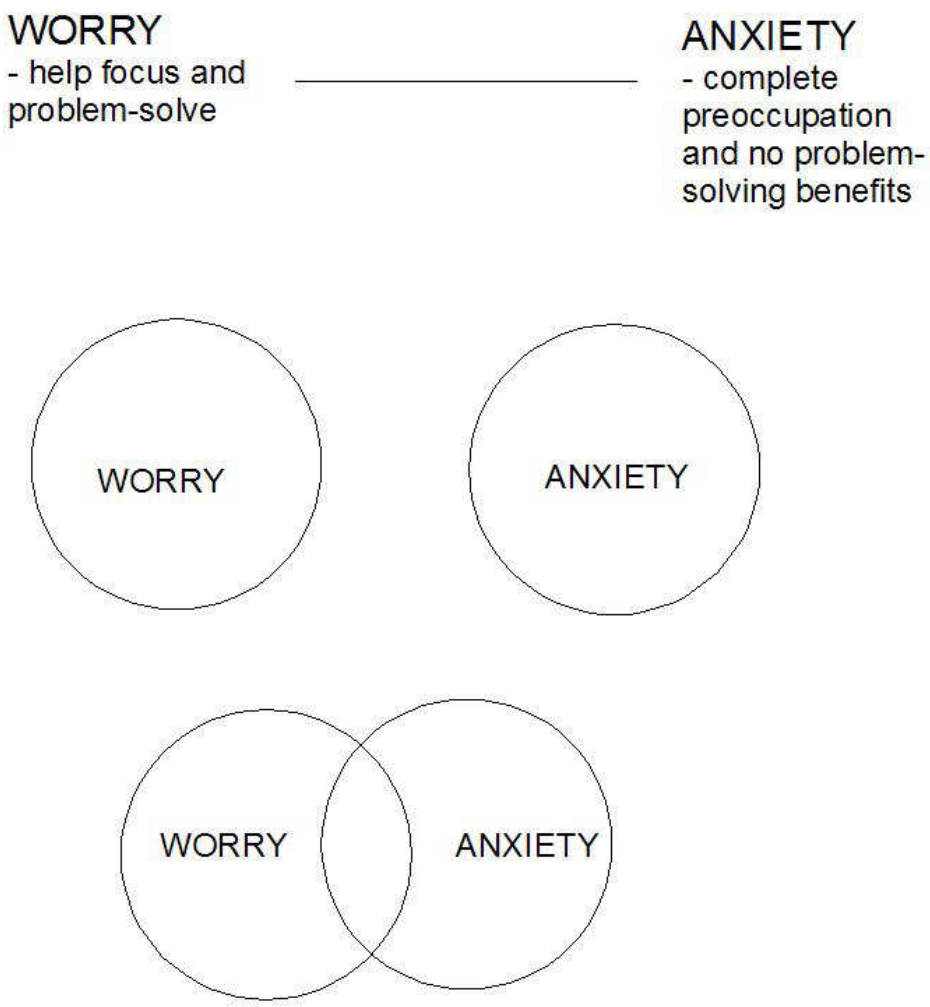

Figure 29.1 - Worry and anxiety a continuum or as separate experiences.

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30. THERAPY THAT CAUSES HARM

Much of the debate about therapy relates to whether it works, and specifically whether individuals improve from it. But there are occasions when individuals will actually get worse from undergoing therapy (figure 30.1). This may be the case for 10% of clients (Jarrett 2008).

NO THERAPY			THERAPY		
↓	↓	↓	↓	↓	↓
No Change	Get Worse	Get Better (Spontaneous improvement)	No Change	Improve	**Get Worse**

Figure 30.1 - Possible outcomes from therapy and no therapy.

There is an assumption that "doing something is always better than doing nothing" [5], but this is not the case (Lilienfeld 2007). There are over 500 "brands" of psychotherapy (Eisner 2000) with many "fad" and "fringe" types that have never been assessed for effectiveness.

Lilienfeld (2007) attempted to compile a list of "potentially harmful therapies" (PHTs). PHTs were operationalised as:

i) Demonstrating harmful psychological or physical effects in clients or others.
ii) Harm includes deterioration and a declined rate of improvement.
iii) Enduring harmful effects.
iv) Harmful effects replicated by individual studies.

From a literature review two levels of PHTs were produced:

a) Level 1 (probable harm) - evidence mainly from randomised controlled trials (RCTs) and meta-analysis of RCTs.

b) Level 2 (possible harm) - evidence mainly from studies other than RCTs.

[5] Sometimes called the "Dodo Bird verdict" (all therapies are approximately equivalent) after the Dodo Bird in "Alice in Wonderland" who said, "Everyone has won, and all must have prizes".

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The level 1 PHTs included:

1. Critical incident stress debriefing (CISD) (appendix 30A) - A single session therapy with individuals after a traumatic event encouraging them to focus on the negative emotions of the event. It is used with the risk of post-traumatic stress disorder (PTSD), but can heighten this risk as not all individuals who experience traumatic events develop the condition.

2. "Scared Straight" programmes - Adolescents with behaviour problems who are at risk of a future life of crime are exposed to the harsh realities of prison life for a day, for example. It was found to increase future arrests compared to no-treatment controls groups.

3. Facilitated communication (FC) - Used to encourage communication through a computer keyboard by non-communicative children with autism. The facilitator holds the child's hands on the keyboard. It has been suggested that facilitators are unknowingly generating the communication.

4. Attachment therapies - For example, holding therapy involves a troubled child being physically held by adults until their anger (from a lack of secure attachment) is released. It involves forceful physical restraint of the child.

5. Recovered-memory techniques - Suggestive techniques, like hypnosis, to recover childhood memories (usually of sexual abuse) have produced false memories of trauma and allegations against family members.

6. Dissociative Identity Disorder (DID)-oriented psychotherapy - The use of techniques to encourage the "alters" of an individual with (DID) ("multiple personalities") to come out to allow for "fusion". There is a risk of the creation of "alters" by suggestion.

7. Grief counselling for individuals with normal bereavement reactions - The risk of increasing depressive symptoms.

8. Expressive-experiential psychotherapies - Techniques that focus on experiencing and releasing powerful

emotions can exacerbate the painful emotions.

9. Boot camp interventions for conduct disorders - Military-style boot camps with strict discipline for adolescents with conduct disorders/behavioural problems. Criminal behaviour higher among attendees.

10. Drug Abuse and Resistance Education (DARE) programmes - Police officers are used to teach children about the risks of drug use ad the ability to say "no" to peer pressure to take drugs. These programmes have been found to be ineffective, or unwittingly encourage alcohol and cigarette use as the emphasis is upon other drugs.

The level 2 PHTs included:

- Peer-group interventions for conduct disorders - Techniques involving peers of individuals with behavioural problems, like group discussion of the behaviour, can exacerbate the problem behaviour.

- Relaxation treatments for panic-prone patients - These can increase anxiety and panic attacks.

ISSUES IN EVALUATING IF THERAPY HARMFUL

There are a number of issues related to establishing if a therapy is detrimental (or beneficial):

i) Definition of "harm". This could include getting better, but at a slower rate than compared to another therapy.

ii) The "file-drawer effect". Studies and clinical trials of therapies that do not find significant improvements tend not to be published, so the negative effects are not known about. For example, Boisvert and Faust (2007) found that nearly one-third of about 200 US practicing psychologists were unaware of negative effects of psychotherapy.

iii) How to establish blame. It is possible that the client who gets worse after starting therapy would have got worse anyway. Is the therapy to blame for that?

iv) How long is enough time. How long should a theory be given before the decision is made that it does not work or is harmful? A demand for instant success is not necessarily helpful nor realistic.

Lambert (2007) suggested the idea of comparing an

individual's progress with similar clients at that state of therapy. This can show whether the client is "on track" or "off track".

v) How much evidence is needed before a therapy is classed as harmful. Randomised clinical trials are the "gold standard" for assessing treatments, but they are far from perfect, and may not actually assess therapy as practiced in the real world (Jarrett 2008).

vi) How results presented. Studies of therapies often present the group means for outcomes, and this can disguise the fact that some individuals improve and others deteriorate. This can be overcome by presenting the results broken down by quartile or using the "number needed to harm" ("the number of patients one would need to expose to the intervention to obtain one harmful outcome compared with the control intervention"; Lilienfeld 2007).

vii) Differences across symptoms. Therapy may aid some symptoms but make others worse, or as some symptoms disappear others appear (eg: dependence on therapist).

viii) Harm to persons other than the client. Some therapies may help the client while causing harm to others around them. For example, an individual in therapy with marital problems may improve by gaining the confidence to divorce, but the spouse suffers as a consequence of this. Or therapy that stimulates recovered memories "could produce a heightened risk of false abuse allegations against family members" (Lilienfeld 2007).

ix) Short-term versus long-term deterioration. Individuals may get worse in the short-term with therapy, but improve in the long-term. How to know if a therapy will produce this ultimate benefit.

x) How to assess client drop-out. Individuals may drop out of therapy (or studies) because they have improved quickly and thus feel better, no longer needing the therapy, or they drop out because of deterioration and dissatisfaction with the therapy.

APPENDIX 30A - CISD

It is designed to prevent PTSD after a traumatic experience. Usually there is a single session lasting 3-4 hours in a group soon after the event (24-72 hours). The focus is the discussion/"processing" of the negative emotions. The assumption being that it is better to get them out in the open as quickly as possible. Furthermore,

the majority of individuals (75%) do not develop PTSD (Sommers and Satel 2005).

In one meta-analysis of RCTs, Litz et al (2002) compared CISD with no treatment or alternative-treatments for the development of PTSD symptoms after traumatic events. The overall effect for CISD was negative (d = -.11) (ie: one-tenth of a standard deviation unit worse PTSD scores than control) (table 30.1).

STUDY	PARTICIPANTS	FINDINGS
Bisson et al (1997)	Hospitalised burn victims: 57 given individual or couple CISD and 46 no treatment	At 13 months: greater PTSD, anxiety, and depression scores in CISD group
Rose et al (1999)	Physical and sexual assault victims: 54 CISD, 52 "psychoeducation", 51 no treatment	At 6 and 11 months: all groups improved and no difference on PTSD and depression measures

Table 30.1 - Two studies included in meta-analysis by Litz et al (2002).

RCTs [6] have found that CISD impedes natural recovery in such situations as fire/burns and motor vehicle accidents (Lilienfeld 2007).

By focusing on negative emotions in one session, CISD may "prime trauma-exposed clients to anticipate PTSD symptoms, thereby creating a self-fulfilling prophecy in which clients passively accept symptoms rather than attempt to overcome them" (Lilienfeld 2007 p65). The use of one session can leave clients with unresolved anxiety that may not have existed at the beginning of the session.

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[6] RCTs difficult to perform because traumatic events are random and so no measure of individuals prior to exposure, and the randomisation to treatment or control groups is seen as unethical (Litz et al 2002).

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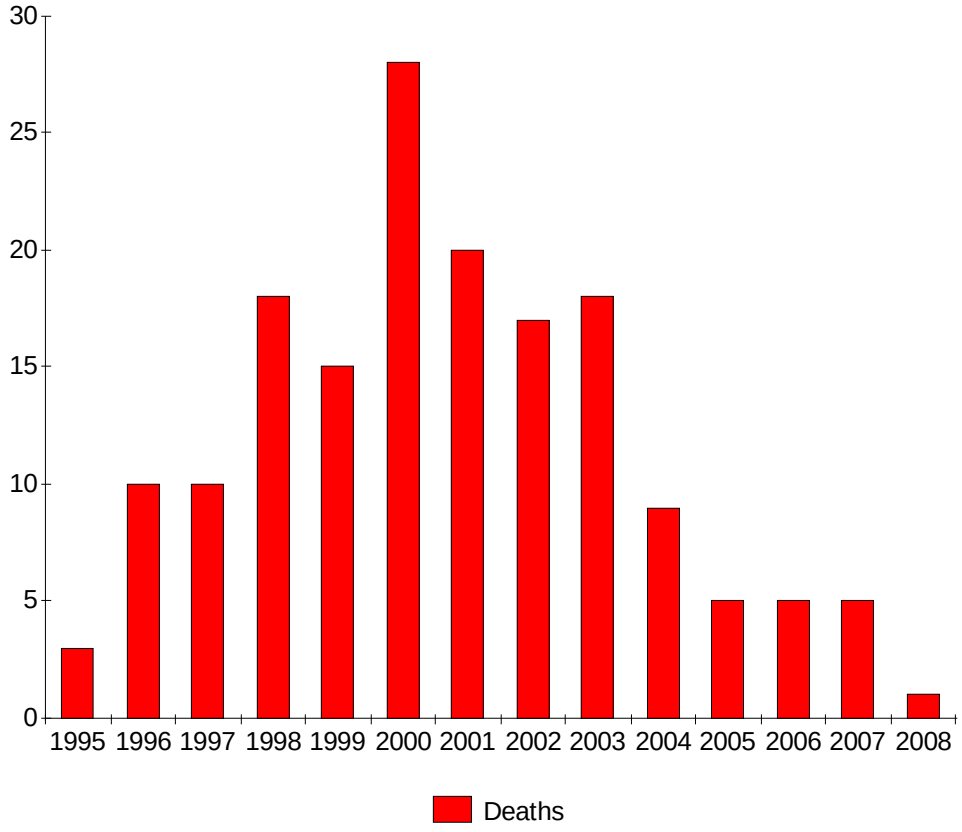
31. AN EPIDEMIC THAT NEVER HAPPENED: CJD IN THE UK

In the 1990s there were mass incinerations of British cattle due to the fear of bovine spongiform encephalitis (BSE) and its transmission to humans as variant Creutzfeldt-Jakob disease (vCJD).

The first death in UK from v CJD occurred in 1995, and in the following ten years just over 150 people died in the UK and thirty elsewhere in the world (Butler 2006). Figure 31.1 shows the officially recorded deaths from vCJD in the UK between 1995-2008.

This is not the epidemic feared (and predicted) in the 1990s. The blood-brain transmission barrier seems to have prevented substances in infected beef products from reaching the brain. Deaths from sporadic CJD (the usual form) remains at one per million people per year worldwide (Butler 2006).



(Source: http://www.cjd.ed.ac.uk/)

Figure 31.1 - Deaths from vCJD in UK.

BSE and CJD are prion diseases [7] caused by the abnormal version of prion protein [8]. The concern was that cows infected with the abnormal prion protein (known as "scrapie prion protein"; Prusiner and Hsiao 1994) would transmit this in their meat to humans. This prion protein, which is resistant to immune system attacks, would travel in the bloodstream to the brain, and there lead to vCJD.

vCJD differs from sporadic CJD most prominently in younger onset. Analysis of the first one hundred cases of vCJD in the UK by the National Surveillance Unit in Edinburgh (Spencer et al 2002) found a mean age of 26 years compared to 50-70 years.

USING THE INTERNET TO STUDY THIS TOPIC

There is plenty of information available on the Internet about this topic, and some helpful and relevant websites are included in this article.

There is also information in Wikipedia. Generally it is felt that this site is not appropriate as the sole basis for academic research (table 31.1), though it can be a good starting point for external links and other websites.

ARGUMENTS FOR	ARGUMENTS AGAINST
1. A lot of information freely available contributed by different individuals (with expertise and knowledge). 2. Incorrect information will be removed by others.	1. There is no guarantee of accuracy, nor that authors are experts, or that incorrect information will be edited. 2. There are no details and names of authors. Academic research and referencing needs an author(s) as the source which can be evaluated.

Table 31.1 - Arguments for and against the use of Wikipedia as the basis of academic research.

1. History of BSE in the UK via BBC Archives

- BSE inquiry (Official UK Government inquiry) (http://news.bbc.co.uk/1/hi/special_report/1999/06/99/bse_inquiry/368776.stm)

[7] Prion disease refers to "proteinaceous infectious agent" (Prusiner 1982).
[8] Prion protein exist naturally in the brain.

- Experience of diary farmer (http://news.bbc.co.uk/1/hi/special_report/1999/06/99/bse_inquiry/358988.stm)

- Government minister (John Gummer) giving beef to his daughter because he said there was no risk (http://news.bbc.co.uk/1/hi/special_report/1999/06/99/bse_inquiry/369625.stm)

2. National CJD Surveillance Unit official site in UK

- (http://www.cjd.ed.ac.uk/)

- Equivalent site in USA (http://www.cjdsurveillance.com/)

3. BSE information

- World Health Organisation factsheet (http://www.who.int/mediacentre/factsheets/fs113/en/)

4. vCJD information

- CDC information in USA (http://www.cdc.gov/ncidod/dvrd/vcjd/)

5. Timeline of events

- New Scientist magazine (http://www.newscientist.com/article/dn9926-timeline-bse-and-vcjd.html_)

- News story timeline (http://newstimeline.googlelabs.com/)

6. Prion

- Diseases (http://www.cdc.gov/ncidod/dvrd/prions/)

- Images of prion protein (http://tinyurl.com/ykv8s37)

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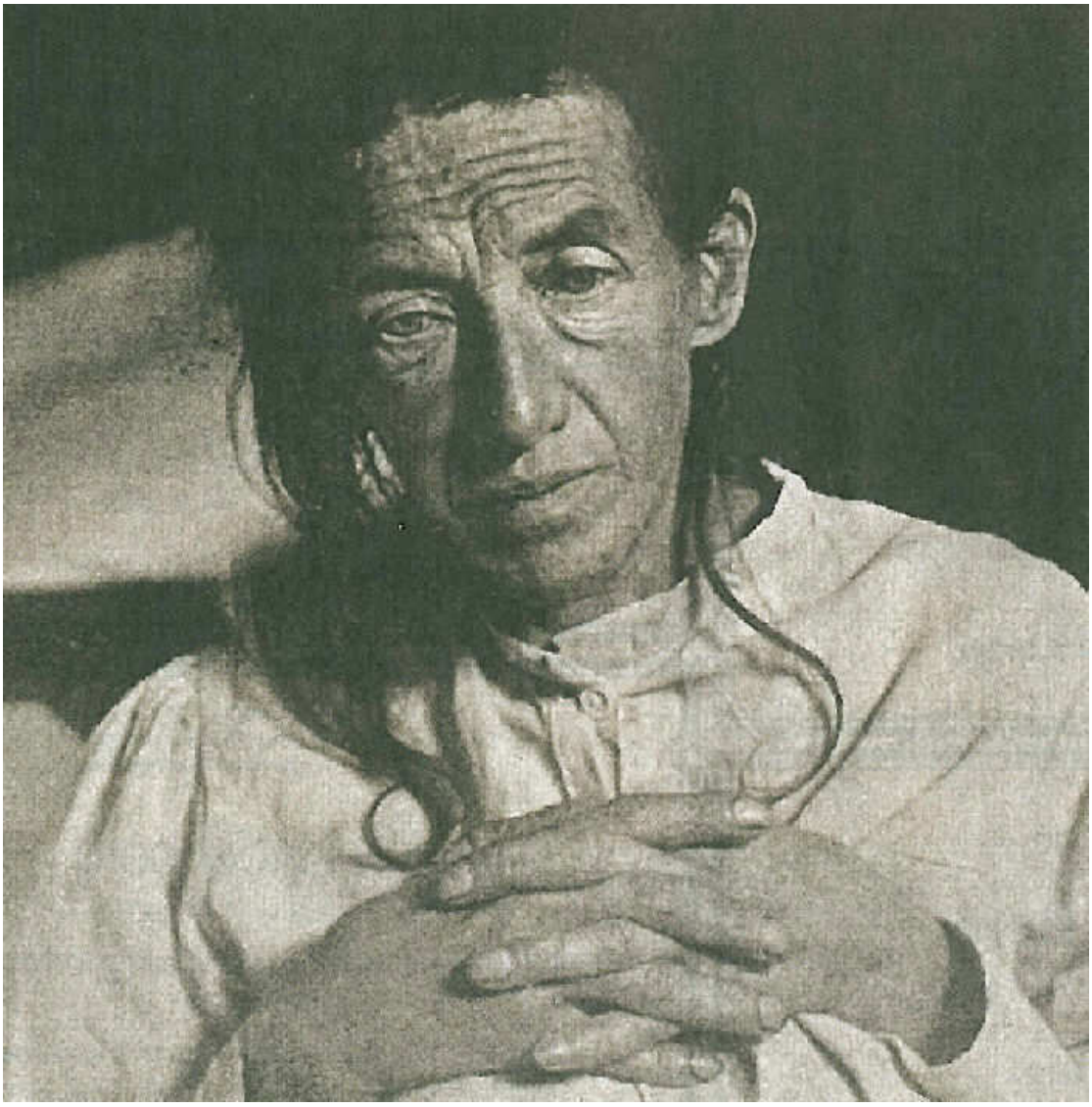
32. DEMENTIA AND ALZHEIMER'S DISEASE: SOME THINGS WE KNOW OR MIGHT KNOW

In a lecture in 1906, Alois Alzheimer described the case of dementia of a woman called "Auguste D" (figure 32.1) in the Hospital for the Mentally Ill and Epileptics, Frankfurt am Main, Germany. This description became known as Alzheimer's disease (AD) or senile dementia of the Alzheimer's type (SDAT) (Maurer et al 1997) (box 32.1).



(Source: Public domain)

Figure 32.1 - Auguste D in 1902.

Auguste D showed the symptoms of dementia at a younger age (51 years old) than was usual, and, after her

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death, it was found that her brain contained clumps outside the cells (plaques of the protein beta-amyloid) and tangles inside the cell (of a protein called tau) (Shurkin 2009).

These plaques and tangles are now seen as the tell-tale sign of AD, but many individuals (eg: 40%) have these in the post-mortem brain without signs of dementia when alive. "Thus, some other pathology, still unknown, might be the cause of Alzheimer's, and the plaques might be either coincidental or a side effect" (Shurkin 2009 p59).

Nov 26, 1901

She sits on the bed with a helpless expression. What is your name? *Auguste*. Last name? *Auguste*. What is your husband's name? *Auguste, I think*. Your husband? *Ah, my husband*. She looks as if she didn't understand the question. Are you married? *To Auguste*. Mrs D? *Yes, yes, Auguste D.* How long have you been here? She seems to be trying to remember. *Three weeks.* What is this? I show her a pencil. *A pen.* A purse and key, diary, cigar are identified correctly. At lunch she eats cauliflower and pork. Asked what she is eating she answers *spinach.* When she was chewing meat and asked what she was doing, she answered *potatoes* and then *horseradish.* When objects are shown to her, she does not remember after a short time which objects have been shown. In between she always speaks about twins. When she is asked to write, she holds the book in such a way that one has the impression that she has a loss in the right visual field. Asked to write Auguste D, she tries to write Mrs and forgets the rest. It is necessary to repeat every word. Amnestic writing disorder. In the evening her spontaneous speech is full of paraphrasic derailments and perseverations.

Extracts from Nov 29, 1901

*. . .*What year is it? *Eighteen hundred.* Are you ill? *Second month.* What are the names of the patients? She answers quickly and correctly. What month is it now? *The 11th.* What is the name of the 11th month? *The last one, if not the last one.* Which one? *I don't know.* What colour is snow? *White.* Soot? *Black.* The sky? *Blue.* Meadows? *Green.* How many fingers do you have? *5.* Eyes? *2.* Legs? *2.*

(Source: Maurer et al 1997)

Box 32.1 - Extracts from Alois Alzheimer's clinical notes on Auguste D.

The mean survival from clinical onset of dementia is 5-9.3 years, and this is lessened by co-morbidity with physical illness, and by being male (Shigeta and Homma 2002). Table 32.1 lists the main stages of cognitive decline (Reed 1991).

STAGE	MEMORY	ACTIVITIES OF DAILY LIVING (ADL)
Forgetfulness	Subjective reports of forgetting	No problems
Borderline AD	Limited objective evidence, but reports from family members of problems	No problems
Mild AD	Objective evidence of memory problems	Little problems
Moderate AD	Objective evidence of dementia; eg: forgetting names of family members	Signs of problems; eg: in choosing appropriate clothes to wear
Moderate severe AD	Clear evidence of dementia; eg: forgetting name of primary caregiver	Semi-dependent
Severe AD	Verbal and motor skills very limited	Totally dependent

Table 32.1 - Stages of cognitive decline in AD.

On a regular basis, stories about dementia and especially AD appear in the media as the findings of academic research are publicised. It can give the impression that progress in knowledge is being made, or, when there are contradictions, that scientists are not really sure. But this is how knowledge and science work. Researchers attempting to piece together information and discover new things. It is like being in the middle of the forest where it is confusing, but from above things are clearer. When we are above the trees, all the different ideas will make sense.

This article is a collection of different ideas about dementia and AD that have appeared recently [9].

DELAYING AND PROTECTING FACTORS

1. The onset of dementia is delayed in individuals with more years of formal education, but once dementia occurs in this group, the memory loss is faster (Goudarzi 2008).

2. Caffeine (the equivalent of two strong cups of coffee per day) staved off and reversed the symptoms of AD in mice (Bee 2009).

3. Protectors against dementia include the Mediterranean-style diet high in fish oil and vegetables (especially

[9] More details of latest research at http://www.alzforum.org/.

blueberries, kale, and broccoli), moderate alcohol intake (1-2 drinks per day), regular exercise, being mentally active, and being married (Bee 2009).

4. A diet rich in omega-3 oils (eg: rapeseed oil) could reduce the risk of AD significantly (eg: 60%), regular fruit and vegetables by 30%, and fish by 35% in French study (Randerson 2007a).

5. Longer leg and arm length in women, and longer arm length in men linked to less risk of AD in a US study that measured limb length of nearly 3000 individuals in their 70s. Limb length is taken as an indicator of nutrition in early life (Sample 2008).

6. Elderly individuals taking statins (LDL-cholesterol-lowering drugs) were less likely to have AD in a post-mortem study of 110 brains in a US study (Randerson 2007b).

7. Marijuana could stave off AD because the active ingredient, tetrahydrocannabinol (THC) blocks the destruction of acetylcholine [10] (Klein 2007).

PREDICTING AND PHYSIOLOGICAL FACTORS

1. Predictors of AD (Zaudig 2002) include:

- Episodic memory problems - subjective complaints over 3-5 years before and complaints of word-finding difficulties before objective tests can detect.
- Apolipoprotein E allele.
- Reduced cerebral glucose metabolism and blood flow.
- Loss of neurons in areas of the cortex and hippocampus (appendix 32A).

Together these factors could show as "mild cognitive impairment" (DSM-IV) - a transitional state between normal ageing and dementia.

2. Early detection of AD is a goal for researchers and practitioners, and new techniques are being developed.

i) Amyloid imaging (Klunk et al 2004) - A radioactive tracer called Pittsburgh compound B (PiB) is injected into the bloodstream, and this binds to amyloid in the brain as seen in a PET scan. Ikonomovic et al (2008) showed a correlation between this technique and

[10] Individuals with AD may have less of this neurotransmitter.

plaque in the post-mortem brain.

ii) Cerebrospinal fluid (CSF) measures (Sunderland et al 2003) - The amount of amyloid and tau proteins in the CSF taken by a spinal tap (via needle inserted into lower back). The less amyloid in the CSF, the more in the brain and the greater risk of AD, while the more tau in the CSF is a risk.

3. The Apolipoprotein E (allele 4) [11] (ApoE4) gene on chromosome 19 is involved in inherited and early onset versions of AD (Al-Khedhairy 2004).

4. The herpes simplex virus-1 (HSV-1) exacerbates effect of AD in mice with ApoE4 gene variant and in cultured brain cells (Geddes 2007).

5. Depression among AD patients is significantly correlated with less income, poorer subjective health, less satisfaction with social contacts, and negative social support (Schulz et al 1992).

TREATMENTS

1. The injection of brain-derived neurotrophic factor (BDNF) into parts of the brain of mice (with a version of AD) led to improvement in memory and learning (Staff 2009).

2. In a double-blind, placebo-controlled study, Winblad et al (2006) reported that donepezil (cholinesterase inhibitor [12]) improved cognition and slowed decline in ninety-five individuals with severe AD.

3. In a systematic review of randomised controlled trials for cholinesterase inhibitors, Kaduszkiewicz et al (2005) could not recommend their use because of methodological weaknesses (eg: no clear duration of study) and small clinical benefits of the studies.

4. Memantine (which blocks NMDA receptor) and donepezil together effective for moderate to severe AD compared to placebo and donepezil (Tariot et al 2004). Over the 24 weeks of the study, two measures of functioning were used - Severe Impairment Battery (SIB) which measures cognitive functioning, and AD Co-operative Study -

[11] An each gene position on the chromosome there are different possibilities (alleles). The most common form here is ApoE3.

[12] Cholinesterase inhibitors slow breakdown of the neurotransmitter, acetylcholine, which is low in AD sufferers (Shurkin 2009). The main types are donepezil, rivastigmine, and galantamine.

Activities of Daily Living Inventory (ADCS-ADL) (measures ability to perform independent activities). On the SIB, the memantine group showed a small improvement compared to a decline in the placebo group between baseline and week 24, and ADCS-ADL decline was less in the former (table 32.2).

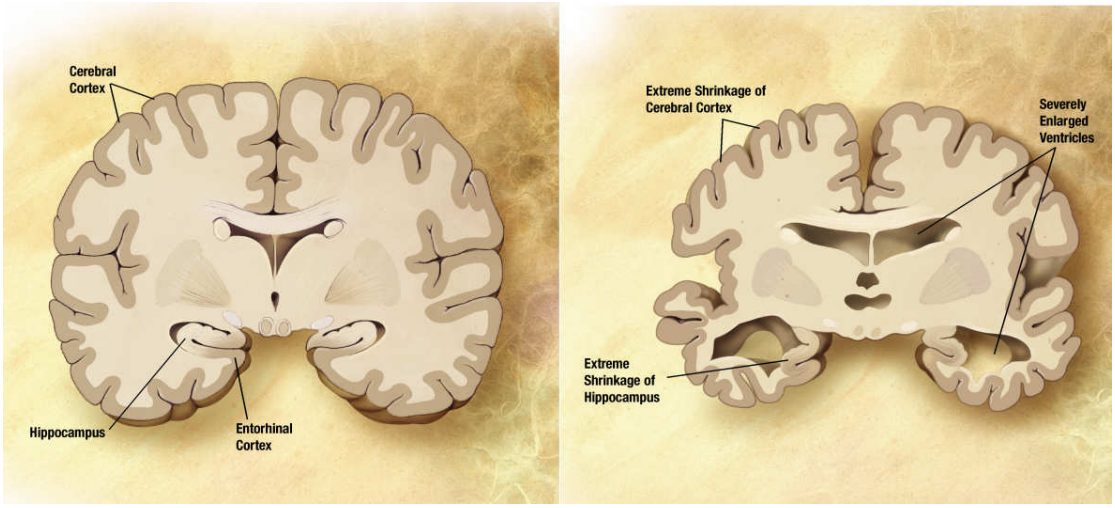
	MEMANTINE		PLACEBO	
	Baseline	Week 24	Baseline	Week 24
SIB *	78.0	79.0	80.0	77.6 ***
ADCS-ADL **	35.5	33.8	35.8	32.5 ****

(* 40 items; score range = 0-100; higher score = better)
(** 19 items; score range = 0-54; higher score = better)
(*** p<0.001; **** p = 0.02)

(After Tariot et al 2004)

Table 32.2 - Mean scores on two measures of functioning.

APPENDIX 32A - BRAIN SLICES



(Source: garrondo; modification of images at http://www.nia.nih.gov/Alzheimers/Resources/HighRes.htm; public domain) [13]

Figure 32.2 - Slice of healthy brain (left) and brain of AD sufferer (right).

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33. SELECTIVE SEROTONIN REUPTAKE INHIBITOR ANTI-DEPRESSANTS AND SIDE EFFECTS

When drugs are tested for effectiveness in clinical trials most of the focus is upon improvements in the condition. In the case of anti-depressants, reductions in depressive symptoms. But there is also the issue of the side effects of the medication. This needs to be part of any risk-benefit analysis of the drug (Healy 2003). This article concentrates on selective serotonin reuptake inhibitor (SSRI) anti-depressants (table 33.1).

TYPE OF SSRI	BRAND NAME
• Citalopram • Fluoxetine • Fluvoxamine • Paroxetine • Sertraline	• Cipramil • Prozac • Feverin • Seroxat (or Paxil) • Lustral (or Zoloft)

Table 33.1 - Types of SSRI anti-depressants.

SUICIDE AND SSRIs

In the USA, 90% of suicides are associated with mental illness, and commonly with depression (Gibbons et al 2007). Most suicides are by individuals not taking anti-depressants, and anti-depressants do reduce the risk of suicide. At the same time, some anti-depressants increase the risk of suicide in depressed individuals.

This has led to a warning in the USA in October 2003 from the Food and Drug Administration (FDA) regarding anti-depressants and youth suicides. The Medicines and Healthcare Products Regulatory Agency in the UK issued a similar warning in December 2003 as did the European Medicines Agency (Gibbons et al 2007).

The upshot is a reduction in prescriptions for anti-depressants, particularly for adolescents, and consequently an increase in suicide by individuals with untreated depression. Gibbons et al (2007) analysed data from the USA and the Netherlands for 2003-5 on adolescent prescriptions of SSRIs, and for suicide rates between 1988 and 2005. A comparison group of US adults sixty years and above, where SSRIs continued to be prescribed, was included.

SSRI prescriptions to adolescents declined by about 20% after the warnings by medical authorities, and the suicide rate increased by over 10%. "If the FDA's conclusion that there may be a causal link between suicide and anti-depressants ..were correct, we would

have expected to see decreases in the suicide rate during the period of declining SSRI prescription rates, but instead we saw an increase in suicide rates, and the increase was greatest in the age range most affected by the decline in SSRI prescription rates" (Gibbons et al 2007 p1360).

Rubino et al (2007) used the UK General Practice Research Database to estimate suicide rates among adults (18-89 years) between different anti-depressants from 1995 to 2005. Citalopram had a completed suicide incidence rate of 0.26 per 1000 person years, and fluoxetine 0.23. This was less than venlafaxine (serotonin and noradrenaline reuptake inhibitor; SNRI) and a tricylic anti-depressant.

Healy (2003) focused on SSRIs and the concern that suicidal ideation and intention are a side effect risk. More importantly, that the manufacturers of these drugs were aware of these risks: "A large number of documents now in the public domain show clinical monitors in Lilly, Pfizer and GSK linking suicidality to their drug. These linkages in fact extend back to the early 1980s at least and to studies in healthy volunteers" (Healy 2003). He concluded, from data in the public domain, that the odds ratio for suicide on a SSRI is 2.5 compared to a placebo (table 33.2).

DRUG	SUICIDE/SUICIDE ACTS (%)	SOURCE
Prozac (comparator)	0.91 (0.65 *)	Company submission to German regulator in 1986
Zoloft/Lustral (comparator/placebo)	0.44 (0.17/0.64)	Company data given to FDA in USA
Seroxat/Paxil (comparator/placebo)	1.52 (1.30/1.44 **)	Company data given to FDA in USA

(* 0.16% Healy re-calculation to include all patients who entered study; ** 0.20% Healy re-calculation)

(After Healy 2003)

Table 33.2 - Rates of suicide for three SSRIs.

The fact that pharmaceutical companies knew that there was such risks has become a legal issue in the USA, in particular, where GSK (GlaxoSmithKline) faced court action from families of children on Seroxat who committed

suicide [14].

As to whether the pharmaceutical companies deliberately withheld such information is open to debate, and to the court cases, but it is the acceptance of the normality of side effects by practitioners that is an issue for me. Extreme side effects, like suicide, are very rare, but there are many more common side effects which are accepted as the way it is. On put another way, you are so ill that "poisoning" you to make you better is alright. A price worth paying.

Table 33.3 lists the side effects of two SSRIs as listed in the official guide for practitioners ("British National Formulary"; BNF) produced by the Royal Pharmaceutical Society of Great Britain.

SSRI	SIDE EFFECTS
General	Gastro-intestinal effects (eg: vomiting, nausea, diarrhoea), weight loss, hypersensitivity reactions (eg: rash), dry mouth, headaches
Fluoxetine	Also changes in blood sugar, fever, neuroleptic malignant syndrome-like event
Paroxetine	Also yawning, and less common ones (eg: panic attacks, arrhythmias)

Table 33.3 - Main side effects reported in BNF 50 (BMA 2005).

Edwards and Anderson's (1999) meta-analysis of comparative studies of the SSRIs found no difference in efficacy between them, but other differences were evident:

- Citalopram - Greatest risk of fatality from overdose;
- Fluoxetine - Slowest onset; more agitation, weight loss, and rashes than others;
- Fluvoxamine - More individuals stopped taking; more gastro-intestinal symptoms than others;
- Paroxetine - More reactions including sweating, and sexual dysfunction than others;
- Sertraline - Less individuals stopped taking than others.

The attitudes of practitioners and the pharmaceutical companies is viewed negatively by many

[14] BBC TV made two programmes about Seroxat in the "Panorama" series (details of 2003 programme at http://news.bbc.co.uk/1/hi/programmes/panorama/2982797.stm and 2007 programme at http://news.bbc.co.uk/1/hi/health/6308871.stm.

people as summed up by Millie Kieve, founder of APRIL (Adverse Psychiatric Reactions Information Line) [15] : "There is reason to be suspicious. When drugs are subjected to clinical trials, not all the findings are disclosed. And because trials are relatively small and short, rarer side effects - and certainly withdrawal effects - go unrecorded" (Kieve 2007).

Turner et al (2008) reported that 94% of trials of anti-depressants were positive for the drug tested, but this was only 51% when unpublished studies that had been given to the FDA were included (table 33.4). The researchers admitted: "We cannot determine whether the bias observed resulted from a failure to submit manuscripts on the part of authors and sponsors, from decisions by journal editors and reviewers not to publish, or both" (p252).

74 studies given to FDA and rated as:

- Positive results for drug (n = 38) - 37 published, 1 not.
- Negative results for drug (n = 36) - 3 published, 22 not; 11 published as positive.

Table 33.4 - FDA-registered studies on anti-depressants and publication.

The justification of side effects is that the drugs work. Kirsch et al's (2008) meta-analysis found that the benefits of fluoxetine and paroxetine are not that large compared to a placebo. The benefits were related to the baseline severity of the depression - "efficacy reaches clinical significance only in trials involving the most extremely depressed patients, and that this pattern is due to a decrease in the response to placebo rather than an increase in the response to medication" (pp0265-0266).

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34. DOES PSYCHOTHERAPY WORK?

Whether psychotherapy works is a big question with no simple answer. This is because it is difficult to test, let alone defining what is meant by "work" (table 34.1).

The most straightforward way is to compare a group of people in therapy with those not to see who improves. This has been done many times, but there are problems with such studies - some are methodologically sounder than others. So not all studies are equal.

- Defining and measuring recovery.
- Different between therapists who use same technique (ie: "good"/"bad" therapists).
- Difference between clients (ie: some individuals more motivated than others).
- Spontaneous recovery (some individuals get better without therapy).
- What is happening outside the therapy to the client.

Table 34.1 - Main problems of trying to establish if therapy works compared to no therapy.

Chambless and Hollon (1998) (C&H) proposed criteria for assessing a treatment as efficacious:

i) The treatment is statistically superior to no treatment or a placebo in randomised controlled trials (RCTs).

ii) The treatment is at least equivalent to alternative "bona fide" treatments in RCTs.

iii) The RCTs involved sound methodology (eg: specific diagnostic groups, standardised techniques of therapy).

iv) The superiority of the treatment demonstrated in at least two independent studies.

When there are a number of studies on a particular topic, researchers attempt to summarise the patterns within them. In terms of the effectiveness of psychotherapy, Eysenck (1952) drew together studies available at the time on psychoanalysis (5 studies) and "eclectic" (mixed) therapies (19 studies). He calculated that 44% of individuals were cured, much improved or improved [16] with psychoanalysis and 64% with eclectic

[16] Recovery was defined as "(i) return to work, and ability to carry on well in economic adjustments for at least five years; (ii) complaint of no further or very slight difficulties; (iii) making successful social adjustment" (Eysenck 1952).

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therapies. This compared with 72% of the control group (individuals hospitalised or "treated" by GPs with sedatives, tonics, and reassurance).

This negative conclusion for psychoanalysis that individuals were better off without it began "an explosion of research into the effects of psychotherapy" (Gross 1990). Subsequent research has become more sophisticated using meta-analysis (eg: Smith et al 1980). This re-analyses the different studies to produce standardised effect sizes. However, it is still dependent on the quality of the individual studies.

Connolly Gibbons et al (2008) applied the C&H criteria to "dynamic psychotherapy". This term covered all forms of therapy that are directly or indirectly related to the ideas of Sigmund Freud. So, it includes traditional Freudian psychoanalysis through to modern brief dynamic therapies. In an ideal world, each form of psychotherapy would be evaluated separately.

Connolly Gibbons et al (2008) evaluated dynamic psychotherapy for different mental disorders:

- Major depressive disorder - The researchers found no recent studies (in last decade) that met the C&H criteria showing psychotherapy to be more effective than no treatment. However, a number of the studies did suggest its effectiveness, particularly as combined with anti-depressants and for a set period of time (eg: sixteen sessions).

- Generalised anxiety disorder (GAD) - "There is no reliable evidence in the empirical literature to support or deny the efficacy of dynamic psychotherapy for GAD" (Connolly Gibbons et al 2008 p99).

- Social anxiety, obsessive-compulsive disorder, agoraphobia, post-traumatic stress disorder, schizophrenia - No relevant recent RCTs.

- Panic disorder - The C&H criteria were met here with a study by Milrod et al (2007). Panic-focused psychodynamic psychotherapy twice weekly for twelve weeks reduced panic attacks for nearly three-quarters of the group compared to improvements for only 39% of the control group (performing applied relaxation).

- Borderline personality disorder - Though studies do not meet the C&H criteria, there is evidence that dynamic psychotherapy is "positively efficacious" here. This means giving the idea further consideration and research.

- Substance abuse - Dynamic psychotherapy was found to

fulfil the C&H criteria for opiate dependence with one study (Woody et al 1987).

Many therapies do not meet the C&H criteria, often because RCTs are not traditionally used in the "therapy world" as in the "medical world" of drug treatments. Does this mean that therapies that fail these criteria are not effective for helping individuals with different mental disorders?

It has been argued that some therapies are better assessed in investigations that link process to outcome (ie: process research, or process-outcome research) rather than the simple focus on outcome in RCTs.

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35. DOES CANNABIS USE CAUSE PSYCHOSIS? HOW TO ANSWER THIS QUESTION

"There is little dispute that cannabis intoxication can lead to acute transient psychotic episodes in some individuals.. and that it can produce shot-term exacerbation of recurrences of pre-existing psychotic symptoms" (Arseneault et al 2004).

But there is a debate around whether cannabis use actually causes (box 35.1) psychosis. This is not an easy topic to establish an accurate answer. Firstly, there is the problem of the nature of the relationship between cannabis use and psychosis.

1. Cannabis causes psychosis - Individuals with no history of or predisposition for psychosis, who would not have developed it otherwise, develop psychosis after cannabis use. This may occur directly, or indirectly through another mechanism. While, for physiological reasons (eg: growing brain), individuals may only be vulnerable during adolescence, for example, and the relationship does not hold for older adults.

2. Cannabis triggers a predisposition for psychosis - Individuals who have a predisposition to develop psychosis have this triggered by cannabis use. A variation is that cannabis use exacerbates psychotic symptoms already present but hidden.

3. Cannabis use and psychosis as a correlation - These two behaviours go together but are not causal. For example, cannabis use and psychosis have a common origin (eg: genetic).

4. Psychosis causes cannabis use - This relationship is considered less, but individuals with psychosis (which may not have shown itself) are inclined to cannabis use (ie: "an attempt to self-medicate"; Ernst 2002).

5. There is no relationship between the two - It is possible that data showing a relationship is an artefact of the method of collection or bias by researchers.

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What is causality? Susser (1991) listed three aspects:
i) Association - The cause and the outcome appear together;
ii) Temporal priority - The cause is present before the outcome;
iii) Direction - Changes in the cause will lead to changes in the outcome.

Association and direction can occur with a correlational relationship, but causality also involves temporal priority. The problem for researchers is in establishing this as the rate of cannabis use is higher among individuals with psychosis.

The waters are muddied further by the findings of Hambrecht and Hafner (1996). Looking back at 232 individuals with schizophrenia, one-third had used drugs at least one year before the onset of the illness, one-third had used drugs and developed the illness within a year, and the remainder used cannabis after the symptoms of schizophrenia had appeared.

Box 35.1 - Establishing causality.

Secondly, there is the question of how much cannabis use is involved. This will vary from once to regular to heavy daily use. Do all levels of use have the same effect or is it only heavy, regular use? How heavy is heavy use?

The remainder of this article looks at the methods used to study whether cannabis use causes psychosis.

RESEARCH METHODS

1. Cross-sectional studies

This is a comparison of two groups - eg: cannabis users versus non-users to see how many show psychosis, or between psychotic and non-psychotic individuals to see how many have used cannabis in the past.

2. Longitudinal studies

This type of study involves following groups of individuals over time to see what happens. In a prospective longitudinal study, a sample of individuals are followed from baseline to see who uses cannabis and who develops psychosis.

3. Cross-cultural studies

This involves a comparison of groups in different societies or cultures. For example, different rates of cannabis use in different countries will lead to different levels of psychosis if there is a link.

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4. Experiments

Experiments allow researchers to control variables by testing humans in laboratory conditions. In reality, it is unlikely to happen for ethical reasons - ie: giving participants cannabis to deliberately make them psychotic.

5. Animal studies

Experiments that control the dose and frequency of cannabis given to non-human animals can show the exact consequences of it.

Table 35.1 summarises the main strengths and weaknesses of the methods, and table 35.2 gives the general problems of researching cannabis use and psychosis.

EXAMPLE OF STUDY - HENQUET ET AL (2005)

This study used data collected as part of the Early Developmental Stages of Psychopathology (EDSP) study, which followed a random representative sample of 14-24 year-olds in Munich, Germany between 1995 and 1999 (n = 2437).

At baseline and follow-up (four years later) assessment was made of fifteen core psychotic symptoms. Cannabis use was defined as five or more in the study period, and frequency rating during heaviest use varied from "almost daily" to "less than once a month".

At follow-up, 17.4% of individuals (n = 424) had experienced at least one psychotic symptom in their lifetime. Overall, 13.1% (n = 320) of the sample admitted to cannabis use at baseline and 14.8% (n = 361) at follow-up. Any cannabis use at baseline increased the risk of psychotic symptom(s). Nearly 20% of individuals with cannabis use reported at least one symptom (adjusted odds ratio 1.53) (figure 35.1). But the odds ratio was 2.23 for "almost daily" cannabis use (figure 35.2). Baseline psychotic symptoms did not predict cannabis use at follow-up, which challenged the idea of self-medication.

METHOD	STRENGTHS	WEAKNESSES
Cross-sectional studies	1. Able to show the difference between groups. 2. Better controls over confounding variables and make-up of groups.	1. Cannot establish causality. 2. Often it is retrospective; eg; psychotic vs non-psychotic and who had used cannabis. There is always the risk of inaccurate recall of information.
Longitudinal studies	1. Able to follow behaviour over a long period of time. 2. Gives researchers a better opportunity to establish causality.	1. Time consuming, and prone to drop out of participants. 2. Cannot control for what else is happening during the study (eg: excessive stress which could cause psychosis)
Cross-cultural studies	1. Show if relationship is universal. 2. Comparison of cross-cultural secondary data can be cheap and quick.	1. Some cultures and societies are not comparable with too many variables, particularly with secondary data. 2. If the study is carried out, it is time-consuming and expensive.
Experiments	1. Best way to establish causality. 2. Researchers can control dose quantity and frequency.	1. Unethical. 2. Only short-term studies are possible that show effects on memory, say, after small and/or single doses.
Animal studies	1. Best way of controlling variables and establishing causality with experiments. 2. Able to test in a way unacceptable with humans (eg: toxic dose).	1. How applicable are the results to humans. 2. Ethics of treating animals in such ways.

Table 35.1 - Main strengths and weaknesses of research methods used to study cannabis use and psychosis.

- Honesty of self-reports about the use of what is an illegal substance.
- Who is willing or not to participate in a study may bias the sample.
- The use of "captive populations" (eg: individuals in psychiatric hospitals) are not representative of the general population.
- Small sample sizes in some cases.
- Lack of information about genetic predisposition to psychosis.
- Limited controls over confounding variables, like everyday stressors.

Table 35.2 - General problems of researching cannabis use and psychosis.

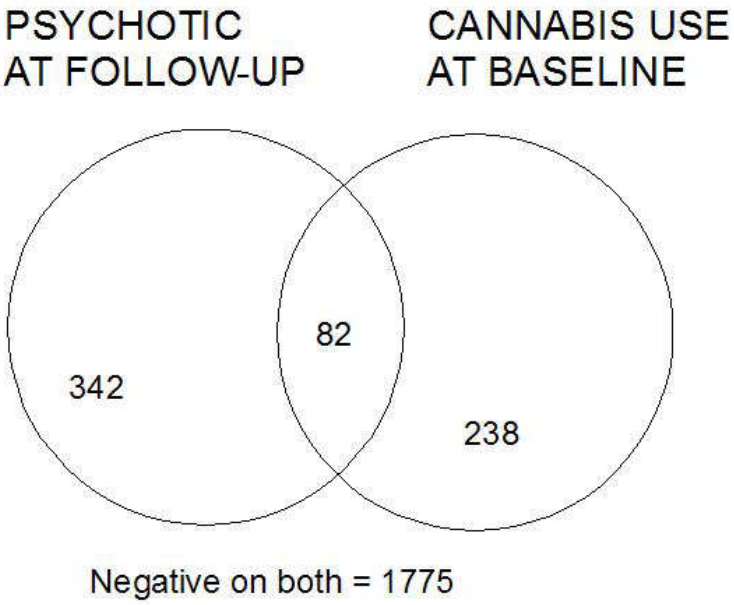

Figure 35.1 - Numbers using cannabis and showing psychotic symptoms.

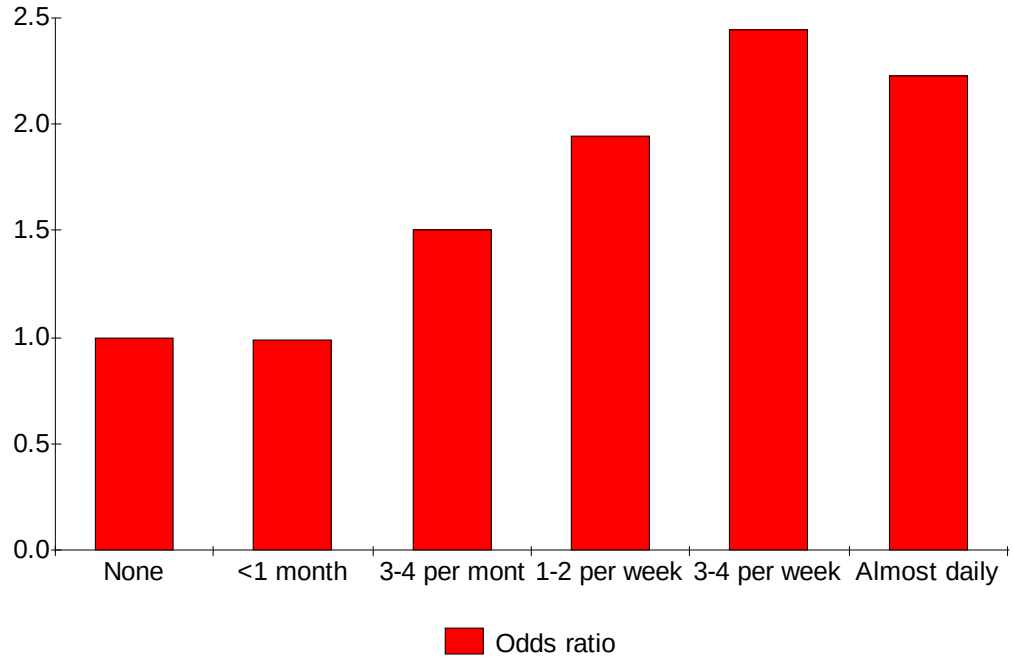

Figure 35.2 - Adjusted odds ratio of psychotic symptoms based on frequency of cannabis use.

CONCLUSIONS

Arseneault et al (2004) found five "good quality" prospective studies based on four samples in Sweden, The Netherlands, and New Zealand. Cannabis use at a certain age was measured and then the amount of psychosis a few years later (table 35.3). The researchers concluded that cannabis use increased the risk of later schizophrenia two-fold, but cannabis use was not the only cause nor always for psychosis to develop.

COUNTRY OF STUDY	AGE OF CANNABIS USE	AGE OF FOLLOW-UP FOR PSYCHOSIS
Sweden	18	33 & 45
The Netherlands	18-64	3 years later
New Zealand	15-18	26-29

(After Arseneault et al 2004)

Table 35.3 - Ages of participants in three studies.

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36. NICOTINE AND COGNITION, SMOKING AND THE BRAIN

Smoking, or more correctly nicotine, has an affect on cognition. it can be both positive and negative. Nicotine is a stimulant as a substance, while smokers are addicts in terms of categorisation. The first example of an experiment relates to the former, and the second example is how the latter is studied.

RUSTED AND TRAWLEY (2008)

Rusted and Trawley (2008) investigated the effect of nicotine on a particular aspect of memory - prospective memory (ProM). This is the use of memory to remember to do something in the future, like remember to post a letter. The basic principle is that smoking after a short abstinence (two hours) improved ProM for smokers.

Rusted and Trawley recruited 32 habitual smokers (5-15 cigarettes per day) and thirty-three "never-smoked" individuals at the University of Sussex, UK. Smokers were asked to abstain for two hours before the experiment and this was checked with a CO smokerlyser (leading to the exclusion of two smokers from the study). All participants were volunteers.

ProM was tested using a lexical decision task (LDT) with an embedded ProM task. This involved remembering certain information while concentrating on another task. The task was simply to respond on a computer keyboard "yes" or "no" if the letters presented on the screen for 500msecs were words (as opposed to random letter strings). There were 192 trials. The ProM aspect was to respond if the letters shown contained "P" or "Q" (set to appear four times each).

At the same time, half of the participants had to listen to an audio stream of digits presented at one digit every two seconds and respond when the number "9" was mentioned. This was known as the working memory load condition.

Another layer was added to the experiment in that the participants also received a nasal spray (1mg) beforehand of nicotine or placebo. This was double blind in that the volunteers nor experimenters administering the test knew which spray was given to whom. Altogether there were eight conditions with approximately eight participants in each group (table 36.1).

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	SMOKER	SMOKER	NON-SMOKER	NON-SMOKER
SPRAY:	NICOTINE	PLACEBO	NICOTINE	PLACEBO
Working memory load	1	2	3	4
No load	5	6	7	8

Table 36.1 - Eight conditions in the experiment.

The score for ProM was the number of letters correctly noticed (out of 8). Other scores for the LDT and the working memory load condition were recorded.

The nicotine spray produced a better performance than the placebo in the no load conditions of the experiment: mean score of 6.1 for conditions 5 and 7 together versus 4.8 for conditions 6 and 8 together in table . But performance was poorer with the nicotine spray in the working memory load conditions: mean score of 3.4 in conditions 1 and 3 together versus 5.0 in conditions 2 and 4 together in table .

So nicotine improves performance for both smokers and non-smokers on a simple task, but not so when tasks are involving a high degree of attention and cognitive resources.

CHIU ET AL (2008)

Chiu et al (2008) recruited 31 smokers in the Houston area of Texas and 31 non-smokers. The experimental task was a gambling game based on the stock market which individuals played while in a functional magnetic resonance imaging scanner. Smokers played once after a normal day's smoking (sated) and once after abstinence for eleven hours (unsated). The order of these days was counterbalanced.

In the "Market Task", participants were given $100 which they could invest, and after half of twenty trials they were told if they had won or lost money ("Live" version) and in the other trials not until the end ("Not Live" version).

The researchers were interested in how the participants would respond to reward expectations, rewards gained, and the difference between what they could have gained and actually did (fictive outcome). It was hypothesises that addicted individuals pursue their addictions irrelevant of negative outcomes and positive outcomes foregone, and so are less influenced by potential consequences in their decision-making.

For example, it is raining outside and a long way to walk to the shop. While the individual is warm indoors

with friends. If this scenario was a non-addict for ice cream, say, a decision would assess the positives and negatives, and probably decide to forego the ice cream. But the addict's decision will not be influenced by these factors, merely the need for cigarettes.

In the experiment, the non-smokers were more influenced by fictive outcomes in their next decisions as well as showing brain responses to this, while smokers (sated/unsated) were not. The non-smokers' brains were more active in certain areas as they tried to predict the next bet. The researchers concluded: "In smokers, higher-order control signals modulating the influence of fictive outcomes on behavioural choice may be impaired, leaving smokers guided only by immediate or experiential rewards and uninfluenced by fictive learning signals".

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37. CHILDBIRTH AND MENTAL ILLNESS

POST-NATAL DEPRESSION

Many mothers experience "baby blues" after the child's birth, but this disappears after a few hours or days. Negative feelings that remain for weeks and months are post-natal or post-partum depression (Gaschler 2008).

Dietz et al (2007) found in the USA that more than half of women (54.2%) with post-natal depression had been depressed before and during the pregnancy (figure 37.1). They used data from a healthcare insurance organisation in Oregon and Washington states, and all live births 1998-2001. This produced 4398 mothers with rates of 10.4% for post-natal depression (up to 39 weeks after birth), 8.7% pre-pregnancy (up to 39 weeks before pregnancy), and 6.9% during pregnancy.

The existence of depression before pregnancy plays a role in post-natal depression along with the hormonal changes after birth: a fifty-fold drop in oestrogen and progesterone (Gaschler 2007). Bloch et al (2000) simulated these changes in sixteen women, and only those who had a history of post-natal depression showed depressive symptoms with the drop in these hormones (5 of 8 vs 0 of 8) (figure 37.2). The women were given an oestrogen and progesterone replacement drug for one month then a placebo for a month (or vice versa). The acute withdrawal of the drug simulated hormones changes after birth.

The average rate of post-natal depression is around 13% of women in studies, mainly from developed countries (O'Hara and Swain 1996). Is this the case for women in poorer countries?

For example, Patel et al (2002) found that 23% (n = 59) of 252 mothers in Goa, India (figure 37.3) could be categorised as depressed at 6-8 weeks after childbirth, and 34 of these were still depressed at six months after birth. The risk of depression was increased by hunger caused by poverty, experience of marital violence, and unhappiness about the child's gender especially if the infant was female. Whereas if the child was male, and the woman already had a female child, the risk of depression was lessened. "The preference for a male child is deeply rooted in Indian society; such gender bias and the limited control a woman has over her reproductive health may make pregnancy a stressful experience for some women. Thus, women who already have a female child face greater stress because of their wish that their new infant be a boy" (p46).

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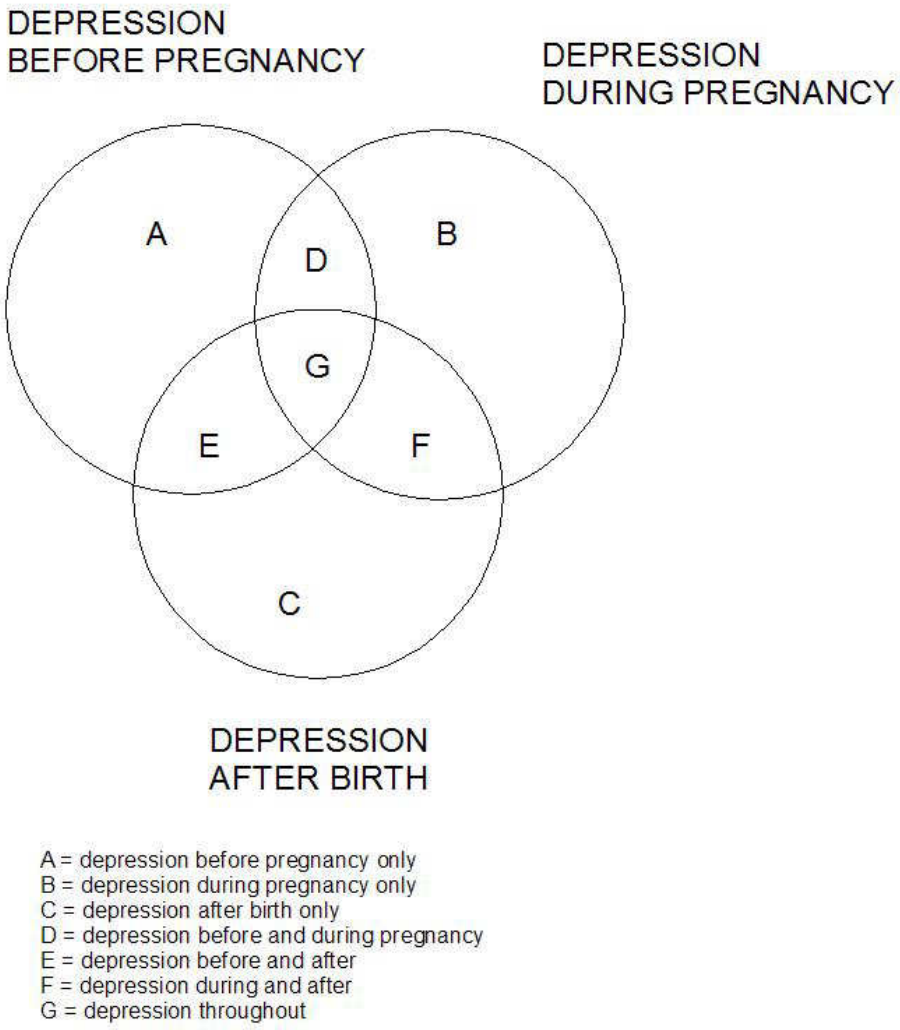
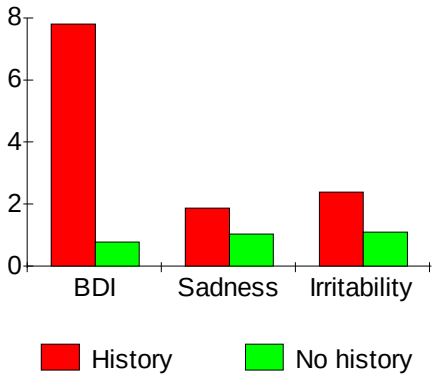


Figure 37.1 - Depression before, during, and after pregnancy.

While in the Tamil Nadu area of India (figure 37.3), Chandran et al (2002) recorded a level of 11% for women with no previous history of depression. The risk factors were low income, birth of daughter when son desired, relationship problems with parents and/or in-laws, stressful life events during pregnancy, and lack of post-natal physical help at home.



(History = women with history of post-natal depression; BDI = Beck Depression Inventory)

Figure 37.2 - Mean scores at oestrogen and progesterone withdrawal.

POST-NATAL PSYCHOSIS

An even smaller number of women suffer from post-natal psychosis. This may or may not be related to pre-pregnancy psychosis.

Harlow et al (2007) used data from the Swedish Medical Birth Register and the Swedish Hospital Discharge Register to identify all women giving birth between 1987 and 2001, and who of those were hospitalised for psychotic illnesses. Each Swedish resident has a unique national registration number which allowed the researchers to compare the women on the two registers.

In total, 612 306 women produced first-child live-births in the study period. Hospitalisation for mental illness before birth was categorised as (i) no pre-pregnancy or pre-natal psychiatric hospitalisation, (ii) hospitalisation for non-psychotic conditions, and (iii) hospitalisation for psychotic ones, and linked to post-natal hospitalisation for psychosis within ninety days of birth.

Overall, hospitalisation for post-natal psychosis was 0.07% of the whole sample. The rate was 0.04% for women with no previous psychiatric hospitalisation and 9.24% of women with a history of hospitalisation (figure 37.4).

(Source: My labels on image in public domain)

Figure 37.3 - Position of Goa and Tamil Nadu states in India.

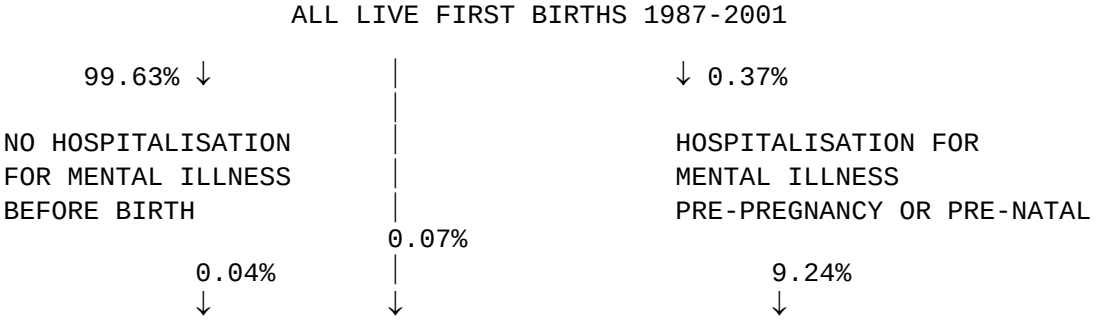


Figure 35.4 - Number of women with post-natal psychosis based on pre-natal psychiatric hospitalisation.

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38. FEAR OF WHAT YOU WANT: TOKOPHOBIA

Tokophobia or fear of childbirth (FOC) [17] is a condition where women are afraid of childbirth despite wanting a baby. At the extreme, sufferers avoid becoming pregnant or have an abortion if become pregnant, while sufferers of a milder form have caesarean sections. One-third of women report some degree of FOC, but only about 10% of the female population are severe (Spice et al 2009).

FOC leading up to birth can produce greater stress during labour with attendant problems (eg: increased pain relief) and post-traumatic stress disorder afterwards with a greater risk of post-natal depression, and delayed bonding with the baby (Hofberg and Brockington 2000) (box 38.1).

FOC stems from a number of other fears including pain during delivery, injury to themselves or the child, death, the unknown, losing control and consequent embarrassment in the delivery room, and lack of support after birth (Spice et al 2009).

Hofberg and Brockington (2000) interviewed over a two-year period 26 women in the West Midlands who had had children. Eight was classed as primary tokophobia where the FOC pre-dates a pregnancy. Contraceptive use was excessive including several methods together. Half the women had had caesarean sections and were positive after the event. The other women had (forced) vaginal deliveries and suffered post-natal depression and some post-traumatic stress disorder symptoms.

Secondary tokophobia (n = 14) developed after a traumatic delivery including severe pain, and fear that they or baby would die. The remaining women in the sample (n = 4) developed FOC as a symptom of depression.

Five women in the sample reported a history of child sexual abuse and three experienced traumatic rape.

Box 38.1 - Hofberg and Brockington (2000).

Spice et al (2009) were interested in the relationship between FOC and anxiety sensitivity (AS). AS is the fear of anxiety-related bodily symptoms (eg: heart palpitations) because it is believed that they are harmful. It is measured by the sixteen-item self-report Anxiety Sensitivity Index (ASI) (Reiss et al 1986), which has three elements:

[17] It was described by Marce in 1858 that "the expectation of unknown pain preoccupies them beyond all measure, and throws them into a state of inexpressible anxiety" (quoted in Hofberg and Brockington 2000).

- AS-physical - Belief that bodily symptoms are signs of physical illness; eg: "when I notice that my heart is beating rapidly, I worry that I might have a heart attack".
- AS-psychological - Belief that they are signs of mental ill; eg: "when I am nervous, I worry that i might be mentally ill".
- AS-social - Fear of public embarrassment of displaying such symptoms of anxiety; eg: "it is important to me not to appear nervous".

FOC was measured by the W-DEQ (Wijma et al 1998). Spice et al approached 110 women in their last four months of pregnancy in Saskatchewan, Canada. It was found that for women with their first child, FOC was predicted by high scores on the AS-physical items of the ASI, and higher levels of general anxiety in life (trait anxiety).

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39. PERFECTIONISM

Perfectionism is "the tendency to set high standards and employ overly critical self-evaluations" (Frost and Marten 1990 p559), and "the disposition to regard anything short of perfection as unacceptable, with perfection defined as flawlessness or an unsurpassable degree of accuracy or excellence" (Stoeber and Kersting 2007 p1094).

Perfectionism as a characteristic can produce a drive to succeed on the positive side, but also lead to easy discouragement and failure to complete tasks on the negative side (Laber-Warren 2009). A theme from the research on perfectionism is that it is the fear of failure that motivates perfectionists, and "new tasks are viewed as opportunities for failure rather than accomplishment" (Frost and Marten 1990). Thus perfectionism has been associated with procrastination (Frost et al 1990).

Perfectionism can be linked to anorexia nervosa, obsessive-compulsive disorder, and depression. Individuals are vulnerable in situations where worth is tied to being the best or doing the perfect job. It can produce self-defeat (Burns 1980).

Higgins' (1987) self-discrepancy theory outlined gaps between the actual and desired behaviours in perfectionism. The self-concept or "actual-self" is the beliefs about actual abilities possessed, and this is viewed in relation to the "ideal-self" (abilities desired) and the "ought-self" (abilities that ought to have). The discrepancy between the actual-self and ideal-self occurs for most individuals, and can be a motivation to change. Perfectionists experience a discrepancy between the actual-self and ought-self as the ought-self and ideal-self are viewed as similar or the same thing. In other words, the perfectionist feels that they ought to be ideal.

For example, in an examination, a perfectionist who gains an exceptionally high mark sees it as the mark they ought to get, if not higher, and does not experience any pleasure from it. Non-perfectionists will gain pleasure from high grades beyond expectations.

Bieling et al (2003) assessed the levels of perfectionism among Canadian undergraduates. Perfectionists tended to aim for higher grades than non-perfectionists, while, in practice, both groups got similar grades. The upshot was that perfectionists continually experienced feelings of failure in terms of never achieving their expectations, while non-perfectionists adapted their expectations to reality

better. The gap between reality and expectations that demotivates individuals is known as the "perfectionism paradox" (Laber-Warren 2009).

Perfectionists can also produce poorer quality work because of the fear of evaluation. Frost and Marten (1990) (appendix 39A) asked female college students to produce a piece of writing under a situation of low or high pressure. High pressure was the evaluation of the work and comparison with others. Students with high perfectionism scores performed poorer in the low pressure and high pressure conditions.

Perfectionism may spring from childhood in a number of ways (Laber-Warren 2009):

- Parents who placed high expectations on their children;
- Imitation by children of perfectionist parents;
- Children of neglectful parents hoping to get noticed by doing everything right;
- Perfectionism as a way of establishing control and order in an unpredictable environment.

In terms of perfectionism as a personality characteristic, Frost et al (1993) found clusters of behaviours. One cluster called "maladaptive evaluation concerns" included being haunted by mistakes, and feeling oppressed by other people's expectations. While "positive striving" included setting high standards, and striving to meet self-imposed goals. The balance of these behaviours influences how the perfectionism manifests itself (ie: positively or negatively). The latter characteristics dominate to produce a "healthy perfectionism" (Laber-Warren 2009).

Healthy perfectionists have high levels of positive characteristics ("perfectionistic strivings") and low levels of "perfectionistic concerns" (negative characteristics), while unhealthy perfectionists have high levels of both. Non-perfectionists have low levels of perfectionistic strivings (Stoeber and Otto 2006).

Hamachek (1978) originally distinguished between "normal perfectionism" and "neurotic perfectionism". With the former type, "individuals enjoy pursuing their perfectionistic strivings", and with the other, "individuals suffer from their perfectionistic strivings" (Stoeber and Otto 2006).

Stoeber and Otto (2006), in a literature review, found that "healthy perfectionist" students responded better to false positive or negative feedback on a test than "negative perfectionists" or non-perfectionists. However, the idea of healthy perfectionism without negative "side effects" is not supported by everyone (Laber-Warren 2009).

In an experiment of attention, perfectionism has been associated with more accurate performance on a visual search task - searching for the presence or absence of a target letter in a string of random letters (Slade et al 1991).

Stoeber and Kersting (2007) compared "perfectionistic strivings" [1] on a number of tasks in Germany. There were reasoning tests (eg: completing a sequence of numbers) and work sample tests (eg; sorting mail). Higher scores on "perfectionistic strivings" significantly correlated with better performance on the tests (table 39.1).

TEST	DETAILS	CORRELATION	PROBABILITY
1. Verbal reasoning	20 incomplete analogies (eg: sheep-wool=bird-?)	.20	p<0.05
2. Numeric reasoning	20 incomplete number sequences (eg: 7,21,18,9,27,24,12,?)	.18	p<0.05
3. Figural reasoning	Choose different view of figure (20 tasks)	.21	p<0.05
Total reasoning score	Total of 1-3 scores	.25	p<0.01
4. Verbal speed test	80 short lists of words and 2 minutes to spot relationships	.18	p<0.05
5. Sorting letters	6 minutes to sort 36 letters	.27	p<0.01
6. Processing emails	8 minutes 30 seconds to sort 42 short emails	.28	p<0.01
Total work sample	Total of 5-6 scores	.32	p<0.001

(After Stoeber and Kersting 2007)

Table 39.1 - Significant positive correlations between "perfectionistic strivings" and test scores [2].

Generally perfectionism is measured by self-reported responses to statements (table 39.2), which is not without problems.

[1] This was measured by response (on six-point scale, "never" to "always") on eight items, like "I strive to be as perfect as possible", and "I have the wish to do everything perfectly".

[2] There were not significant correlations for two speed tests.

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MEASURE	CHARACTERISTICS OF PERFECTIONISM MEASURED	EXAMPLE OF ITEMS
Almost Perfection Scale (APS) (Johnson & Slaney 1996)	Standards	"I try to do my best at everything I do"
Perfectionism Questionnaire (PQ) (Rhéaume et al 2000)	i) Negative consequences of perfectionism ii) Perfectionist tendencies	i) "Everything is spoiled if an imperfection gets by me" ii) "I like things I do to be perfect"
Multidimensional Perfectionism Scale (MPS) (Hewitt & Flett 1991)	i) Self-oriented perfectionism ii) Socially prescribed perfectionism iii) Other-oriented perfectionism	i) "One of my goals is to be perfect in everything I do" ii) "The people around me expect me to succeed in everything I do" iii) "If I ask someone to do something, I expect it to be done flawlessly"

(After Stoeber and Otto 2006)

Table 39.2 - Examples of commonly used self-reported measures of perfectionism.

APPENDIX 39A - FROST AND MARTEN (1990)

106 female students at a small US private college for women were given the Multidimensional Perfectionism Scale (MPS) [3] (Frost et al 1990) to complete. Respondents in the top 25% of scores (perfectionists) and in the bottom 25% (non-perfectionists) were recruited for the study (n = 51).

The participants were randomly assigned to the low-evaluation-threat (LET) or the high-evaluation-threat HET) conditions (table 39.3). In the former, the task was to rewrite a 166-word paragraph from a textbook in their

[3] The MPS is a 35-item questionnaire giving an overall perfectionism score, and five sub-scores:

- Concern over mistakes; eg: "People will probably think less of me if I make a mistake";
- Personal standards; eg: "If I do not set the highest standards for myself I am likely to end up as a second-rate person";
- Parental expectations; eg: "My parents expected excellence of me";
- Doubting of actions; eg: "I usually have doubts about the simple everyday things that I do";
- Parental criticism; eg: "As a child I was punished for doing things less than perfect".

own words. In the HET condition, the participants were given the same task, but told that their work would be evaluated as part of a national competition.

Two college professors rated the quality of rewriting of all participants for the researchers on a scale of 1 to 5. They worked independently and were blind to the participants' condition and MPS score [4].

	HIGH PERFECTIONISM SCORE	LOW PERFECTIONISM SCORE
LOW EVALUATION THREAT	13	13
HIGH EVALUATION THREAT	13	12

Table 39.3 - Four conditions of the experiment and number of participants in each.

The researchers made the following hypotheses:

1 - High perfectionist (HP) students would report more negative feelings [5] before and after the task (ie: performance anxiety) than low perfectionist (LP) students generally. This would be even more so in the HET condition.

2 - HP students would devalue their performance more than LP students after the task was completed (eg: "it's not as good as I could have done").

3 - HP students would produce poorer quality writing as scored by independent judges.

In terms of the findings:

Hypothesis 1 - HP students reported significantly higher negative feelings than LP students generally (mean: 35.56 vs 26.12 out of 70). In the HET condition, the means were 39.3 (HP students) and 24.8 (LP students) ($p<0.05$) (figure 39.1). Prediction supported.

[4] Inter-rater reliability was 71% overall with perfect agreement on 63% of participants, and no disagreement beyond two rating scale units.

[5] Measured by rating 10 emotions (eg: worried, anxious, relaxed) on seven-point scale.

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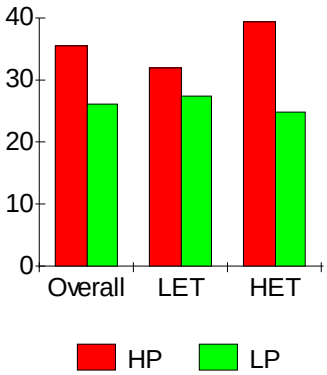

Figure 39.1 - Ratings of negative feelings towards task.

Hypothesis 2 - Generally HP students reported that they should have done better significantly more than LP students (mean: 5.04 vs 4.56 out of 7). Prediction supported.

Hypothesis 3 - Overall, HP students' writing was judged significantly poorer (mean: 3.08 vs 3.60 out of 5). There were not significant differences in the individual conditions. Both types of students produced poorer work in the HET condition. Prediction generally supported.

Overall, HP students differed from LP students on actual task performance and feelings about the task. This was more so when the evaluation of the task was emphasised (HET condition).

Validity

This study like all experiments is faced with a number of issues relating to validity.

1. Internal validity - This refers to "all the possible 'threats' that might exist in an experiment which lead us to think we have demonstrated an effect of a particular independent variable on a particular dependent variable" (Coolican 2001).

Examples of possible "threats" in this study include:

- Ability of participants on task;
- Familiarity with writing task;
- Extraneous noise/distractions during experiment;
- "Demand characteristics" (ie; participants behaving as they believe the experimenter wants them to behave);
- Behaviour of experimenter during interaction with

participants;
- Experimenter effects: expectations of researchers;
- Statistical errors; eg: wrong or inappropriate statistical test.

2. External validity - This refers to the applicability of the findings to other places, people, and times.

- Ecological validity - applicability to other situations, specifically outside the laboratory experiment in a naturalistic or "real life" situation; eg: other colleges in the USA, other countries and cultures;

- Population validity - applicability of results to other sections of society; eg; other age groups, ethnicity, non-students of the same age, other occupations;

- Time - applicability to future and past.

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40. SIMILARITY IN BELIEFS BETWEEN EATING DISORDERS AND OBSESSIVE COMPULSIVE DISORDER

Shafran (2003) identified six areas of similarity in beliefs between individuals with obsessive-compulsive disorder (OCD) and those with eating disorders (ED):

- Perfectionism
- Intolerance of uncertainty
- Overestimation of threat
- Control of thoughts
- Importance of thoughts
- Responsibility (feeling responsible for things, particularly bad, that happen that have no control over).

Lavender et al (2006) investigated this overlap among 177 volunteers from the research register of the Eating Disorders Research Unit (EDRU) at the Institute of Psychiatry, London. The beliefs were measured by different questionnaires:

- Obsessive Beliefs Questionnaire (OBQ) (OCCWG 2001 [6]) - This measures the six areas of belief with 87 statements, like "If I am uncertain, there is something wrong with me" (intolerance of uncertainty), answered with a seven-point Likert scale ("disagree very much" to "agree very much") (box 40.1).

- Interpretation of Intrusions Inventory (III) (OCCWG 2001) - This measures intrusive thoughts with thirty-one statements, like "Thinking this thought could make it happen" (box 40.2). A seven-point response was offered as above.

- Magical Ideation Scale (MIS) (Eckblad and Chapman 1983) - Thirty statements rated as true or false to measure magical thinking (eg: "I think I could learn to read others' minds if I wanted to"; "I sometimes have a feeling of gaining or losing energy when people look at me or touch me"; "Some people can make me aware of them just by thinking about me").

[6] OCCWG = Obsessive Compulsive Cognitions Working Group.

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- Having bad thoughts or urges means I'm likely to act on them.
- Having control over my thoughts is a sign of good character.
- If I don't control my unwanted thoughts, something bad is bound to happen.
- I often think things around me are unsafe.
- There is only one right way to do things.
- Things should be perfect according to my standards.
- If I fail at something, I am a failure as a person.
- Even if harm is unlikely, I should try to prevent it at any cost.
- For me, having bad urges is as bas as actually carrying them out.
- If I don't act when I foreseen danger, than I am to blame for any consequences.
- I would be a better person if I gained more control over my thoughts.
- Things should be perfect according to my own standards.
- I can have no peace of mind as long as i have intrusive thoughts.
- The more I think of something horrible, the greater the risk it will come true.
- When I see nay opportunity to do so, i must act to prevent bad things from happening.
- I must think through the consequences of even my smallest actions.

(After Frost and Steketee 2003)

Box 40.1 - Examples of statements from the OBQ.

- Having this intrusive thought means I'm out of control.
- Having this thought means I am weird or abnormal.
- I would be irresponsible if I ignored this intrusive thought.
- Having this intrusive thought means I am a terrible person.
- I must have control over this thought.
- I'll feel guilty unless I do something about this thought.
- I should not be thinking this kind of thing.
- If I ignore this thought, I could be responsible for serious harm.

(After Frost and Steketee 2003)

Box 40.2 - Examples of statements from the III.

Eating disorders were measured by the Eating Disorder Diagnostic Scale (EDDS) (Stice et al 2000). From this, 52 participants were diagnosed with anorexia nervosa, 42 bulimia nervosa, 6 binge-eating disorder, and twenty-six with eating disorder not otherwise specified. Fifty-one individuals were not given a diagnosis of an ED, but fifteen of them did not complete the EDDS properly. These individuals were excluded along with the six participants with binge-eating disorder leaving 156 participants for analysis (120 with ED [7], 36 without). The

[7] Analysis of data showed no difference on the questionnaires between sufferers of the different types

vast majority (146 - 94%) were female. In fact, there was only one male as nine participants did not give their gender.

The data collected here were compared with that of 248 OCD sufferers and 105 anxiety sufferers (controls) in a study by OCCWG (2003), and 61 OCD sufferers and 86 undergraduate healthy controls in Einstein and Menzies (2004a, 2004b) [8].

The statistical analysis of results found significant differences between the groups on all three questionnaires used.

- OBQ - Current ED sufferers had significantly higher scores on all six areas of belief than recovered ED individuals, OCD sufferers, and anxiety controls, except for on the responsibility sub-scale.
- III - Current ED sufferers had significantly higher scores here as well.
- MIS - As above (table 40.1).

	OBQ (total = 609) *	III (total = 217) **	MIS (total = 30)
Current female ED sufferers (N = 120) ***	393	164	8.76
Recovered female ED sufferers (N = 260 ***	323	113	5.80
OCD sufferers (N = 248) ****	343	154	/
OCD sufferers (N = 61) *****	/	/	6.08
Anxiety controls (N = 105) ****	314	133	/
Undergraduates (N = 86) *****	/	/	4.85

(* combination of six sub-scales; ** combination of three sub-scales; *** Lavender et al (2006); **** OCCWG (2003); ***** Einstein and Menzies (2004a, 2004b))
(After Lavender et al 2006)

Table 40.1 - Summary of the findings by Lavender et al (2006).

of ED.

[8] OCCWG (2003) used OBQ and III, while Einstein and Menzies used MIS.

Evaluation of Lavender et al (2006)

1. The study showed the similarity in beliefs between ED sufferers and OCD sufferers, but also the small differences between them, and between current and recovered ED sufferers.

2. The obsessive-compulsive type beliefs among ED sufferers may represent a co-morbidity of OCD and ED rather than the similarity in beliefs. This study did not distinguish between ED sufferers with a co-morbidity of OCD.

3. The diagnosis of ED was based upon the self-reported EDDS, as were all the other questionnaires. Self-reported questionnaires depend upon the accuracy of recall and honesty of the respondents.

4. The sample was self-selecting both in terms of those on the EDRU research register, and those who returned the questionnaires completed (177 of 400). They may not have been representative of the ED population, and thus had higher or lower scores. Generalisation of the results is consequently limited.

5. The questionnaires used were established commonly-used ones with internal and external reliability, validity, and discriminability.

6. The comparison of data with the other studies has limitations. Lavender et al had to use the data as presented, and had no control over gender, age, or diagnosis as with their sample. It would have been better for lavender et al to have their own comparison samples. Table 40.2 compares the samples.

	Lavender et al (2006)	OCCWG (2003)	Einstein and Menzies (2004a, 2004b)
Mean age (yrs)	35.2	35	33 (OCD) 22 (Undergrads)
Gender (% female)	All	56% (OCD) 68% (anxiety)	56% (OCD) 62% (Undergrad)

Table 40.2 - Age and gender of the three studies.

COGNITIVE DISTORTIONS

Another similarity between OCD and ED is the presence of cognitive distortions which contribute to the maintenance of the disorder, if not the cause.

In the case of OCD, Shafran et al (1996) described "thought-action fusion", which is the belief that intrusive thoughts can influence an actual event (eg: a loved one will die in a car crash). Thus the sufferer becomes responsible to perform rituals to stop the thoughts becoming a reality.

Shafran et al (1999) proposed that for ED the cognitive distortion is "thought-shape fusion" (TSF). I has three components (Shafran and Robinson 2004):

- Likelihood TSF - thinking about eating a forbidden food can lead to weight gain;
- Moral TSF - thinking about eating a forbidden food is morally equivalent to eating it;
- Feeling TSF - thinking about eating a forbidden food leads to feelings of fatness.

Thus, "If I think about overeating or eating prohibited foods, then I think that I may have gained weight/I am immoral/I feel myself as fatter" (Shafran and Robinson 2004 p400).

But TSF is not the seen as delusions (in the clinical sense). These individuals "are fully aware that their thoughts cannot actually influence their weight; however, they then go on to describe an irrational belief that this does occur" (Shafran and Robinson 2004 p406).

Shafran and Robinson (2004) investigated TSF among forty-two women with a clinical ED [9] recruited through Warneford Hospital, Oxford and The Royal Free Hospital, London, and forty-two age-matched advertisement-recruited healthy women.

TSF was measured by the 34-item Thought-Shape Fusion Questionnaire (TSFQ) (Shafran et al 1999). The first half of this questionnaire (17 items) contains statements based on the three components of TSF above (eg: "Just picturing myself gaining weight can really make me gain weight", "If I think about breaking my diet, it is almost as unacceptable as really breaking my diet", "I feel fatter just by thinking about gaining weight") and individuals rated on a scale of 0 ("not at all") to 4 ("totally"). The score of this "concept" section can vary from 0 to 68.

The second half of the questionnaire ("interpretation" section) (17 items) asks about thinking about eating "forbidden" foods with statements like "I am a pig", and "I lack self-discipline". The same rating scale was used.

[9] Diagnosed by DSM-IV criteria, 10 had anorexia, ten bulimia, and the remainder were ED not otherwise specified.

The two scores on the TSFQ were correlated with scores on the Eating Disorder Examination - questionnaire version (EDE-Q) (Fairburn and Beglin 1994) [10], the Body Checking and Avoidance Questionnaire (BCAQ) (Shafran et al 2004) [11], and the Beck Depression Inventory (BDI) (Beck et al 1961) [12]. Pearson's product moment correlation coefficient was the statistical test used (table 40.3).

STRENGTHS	WEAKNESSES
1. A parametric test, which means it is more powerful than the non-parametric equivalent (Spearman's rank correlation coefficient). 2. Robust if parametric assumptions are not met. 3. More sensitive to the data collected than non-parametric tests. 4. Less chance of type I or type II errors (Coolican 1990). 5. Does take into account actual scores whereas Spearman's test ranks data.	1. Parametric tests make assumptions about the distribution of data. 2. Value can be distorted by outliers. 3. Cannot be used with nominal and ordinal data. 4. Complicated to calculate. 5. Curvilinear relationships will produce low correlation scores that are non-significant.

Table 40.3 - Strengths and weaknesses of Pearson's product moment correlation coefficient.

The TSFQ scores significantly correlated with the EDE-Q scores, and the ED sufferers had significantly higher scores on the TSFQ than the controls (table 40.4).

The correlations between the TSFQ scores and the BCAQ and the BDI scores were not significant when controlling for the BDI scores. In other words, for individuals with ED but not depression. There were significant correlations for individuals with ED who were depressed.

[10] Measures frequency of ED behaviours in the preceding 28 days with 36 items and a seven-point rating scale.

[11] Measures the frequency of checking areas of the body (eg: pinching stomach) over last 28 days with 23 items and a six-point scale (0 = "Not at all - not interested" to 4 = "Checked three or more times a day", 5 = "Avoided doing so because of possible distress").

[12] Twenty-one items on the severity of depressive symptoms.

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	ED sufferers	Controls
"Concept" section	1.0 - 1.3 *	0.2
"Interpretation" section	1.7 - 2.0	0.3

(* different scores for three types of ED)

(After Shafran and Robinson 2004)

Table 40.4 - Group mean scores on TSFQ as converted into standardised scores.

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41. METACOGNITION AND ANOREXIA NERVOSA

Cognition refers to the thinking process, and metacognition is knowledge about the thinking process. In other words, metacognition is individuals' awareness of how their thoughts "work".

Wells (2000) described three aspects of metacognition:

i) Metacognitive knowledge - beliefs about cognitions; eg: "worrying thoughts not good";

ii) Metacognitive experiences - assessment of the meaning of cognitions; eg: "worrying thoughts mean I am losing control";

iii) Metacognitive control strategies - methods used to control thoughts; eg: ignore worrying thoughts.

There can be adaptive and maladaptive versions of these three aspects (table 41.1).

	ADAPTIVE	MALADAPTIVE
Knowledge	Not good thought	An accurate picture of reality
Experiences	"I am becoming too negative"	"Only bad things ever happen to me"
Control strategies	"Think positively"	Must use techniques to stop thoughts; eg: endless counting
Result	Awareness of negative thoughts and problem resolved	Ritualistic behaviours as in obsessive-compulsive disorder

Table 41.1 - Aspects of metacognition in relation to thought, "One bad event leads to another and so on".

Applied to anorexia nervosa (AN), metacognitive knowledge is a thought like "If I don't worry about my weight, then I'll get fat". The assessment (metacognitive experiences) is that thought is a positive thing, and a control strategy becomes to mentally check the amount eaten over the course of the day.

Cooper et al (2007) studied such beliefs among sixteen women with a history of AN, fifteen "normal" dieting women, and 17 non-dieting women. They completed the Metacognitions Questionnaire (MCQ) (Wells and

Cartwright-Hatton 2004) (box 41.1) [13].

There are five subscales:

- Positive beliefs - worry as useful coping strategy.

Eg: I need to worry or think about food and my eating, weight and shape in order to remain organised.
A Do not agree
B Agree slightly
C Agree moderately
D Agree very much.

- Uncontrollability and danger - worry as dangerous and uncontrollable.

Eg: My worrying thoughts about food and my eating, weight and shape are not productive

- Cognitive confidence - lack of confidence in memory.

Eg: I don't completely trust my memory for food and my eating, weight and shape.

- Need for control - importance of controlling thoughts, especially worrying one.

Eg: I should be able to control my worries and thoughts about food and my eating, weight and shape.

- Cognitive self-consciousness - awareness of thought processes.

Eg: It is hard for me to distract myself from my worries and thoughts about food and my eating, weight and shape.

(Source: http://weblearn.ox.ac.uk/site/users/lina1242/part2/metacog/)

Box 41.1 - Subscales of MCQ and examples of questions.

There were significant differences between the three groups on the subscales, except positive beliefs, and particularly the AN group differed from the other two groups of women. This meant that anorexic women saw their worrying thoughts as more uncontrollable, had less confidence in their memory, had a greater need to control thoughts, and were more aware of their won thoughts than the other women.

This study was small-scale as it was a preliminary investigation. The sample is less important in such studies because it is accepted that the generalisation of results will be limited (table 41.2).

[13] Copy available to complete at http://weblearn.ox.ac.uk/site/users/lina1242/part2/metacog/.

STRENGTHS	WEAKNESSES
1. All groups of similar mean age: 29.6 years (AN group), 34.0 (dieters), and 26.2 (non-dieters). 2. AN group recruited through therapists. 3. 13 of the AN group had a history of the binge-purge subtype, which is associated with obsessive compulsive disorder. 4. The dieting and non-dieting groups recruited by convenience sample; ie: volunteers from Warneford Hospital, University of Oxford (workplace of researchers). 5. All volunteers.	1. All AN group, but one, had received treatment. 2. Only AN, not other eating disorders. 3. Not all AN group had a current DSM-IV diagnosis. 4. Duration of AN ranged from 1 to 30 years (median = 6 years). 5. Dieting group varied from four weeks to twenty years dieting (median = 26 weeks), but minimum of four weeks was criteria for inclusion.

Table 41.2 - Strengths and weaknesses of the sample studied.

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42. INTERVIEW VERSUS SELF-REPORT QUESTIONNAIRE FOR MEASURING EATING DISORDERS

Questionnaires tend to be interviewer-administered where the interviewer asks the questions and codes the replies, either face-to-face or by telephone, and self-reported. In the latter, the individual completes the questionnaire themselves alone via the Internet or mail. There are strengths and weaknesses to both types (table 42.1).

INTERVIEWER-BASED	SELF-REPORT
Strengths 1. Able to guide interviewees through complex questions and information. 2. Terms and language used can be clarified. 3. Interviewer able to collect information not directly in questions (eg: body language), and control environment (eg: who else present). Weaknesses 1. Costly to use including the training of interviewers. 2. Usually take longer to administer. 3. Interviewer-interviewee variables like embarrassment in admitting to certain behaviours.	Strengths 1. Easier to use anonymously, which reduces the risk of embarrassment for certain questions. 2. Cheaper to use. 3. Quicker and easier to obtain large sample. Weaknesses 1. Nobody to clarify misunderstandings of the questions. 2. No control over who is present during completion and their influence on the respondent. 3. Dependent upon the honesty and accuracy of recall of information (also weakness of interviews).

Table 42.1 - Main strengths and weaknesses of interviewer-based and self-report questionnaire.

The Eating Disorder Examination (EDE) (Cooper and Fairburn 1987) can be used with an interviewer or self-reported. Fairburn and Beglin (1994) recruited thirty-six female patients with anorexia nervosa and bulimia nervosa, and 243 women (aged 16-35 years) in Oxfordshire as controls.

Each individual completed the self-report version and was interviewed within 24 hours. The order was not

counterbalanced to compensate for order effects [14] because it was felt by the researchers that being interviewed first would influence the self-report answers.

The EDE interview (11th edition) measures eating disordered behaviour in the last 28 days, including restriction of food intake to influence weight, frequency of binge eating and self-induced vomiting. Each item is rated on a seven-point scale. The self-report version (EDE-Q) was a simpler version of the above.

In terms of the frequency of three key behaviours:

- Self-induced vomiting - No difference in amount reported for both measures and both samples;
- Laxative misuse [15] - There was a significant difference [16] between the two measures for the controls only;
- Binge eating - Both samples showed significant differences between the two measures (table 42.2).

BEHAVIOUR	PATIENTS	CONTROLS
Self-induced vomiting	EDE > EDE-Q; ns	EDE-Q > EDE; ns
Laxative misuse	EDE > EDE-Q; ns	EDE-Q > EDE; $p<0.05$
Binge eating	EDE-Q > EDE; $p<0.05$	EDE-Q > EDE; $p<0.001$

(ns = not significant)

(After Fairburn and Beglin 1994)

Table 42.2 - Differences on two measures of frequency of three behaviours.

The control group (ie: not diagnosed with eating disorders) reported higher frequencies of these behaviours on the self-report questionnaire. The researchers worried that this may be an over-estimate, particularly for binge eating, because of the misunderstandings of the terms or a problem of definition. Self-induced vomiting, for example, is a less ambiguous term.

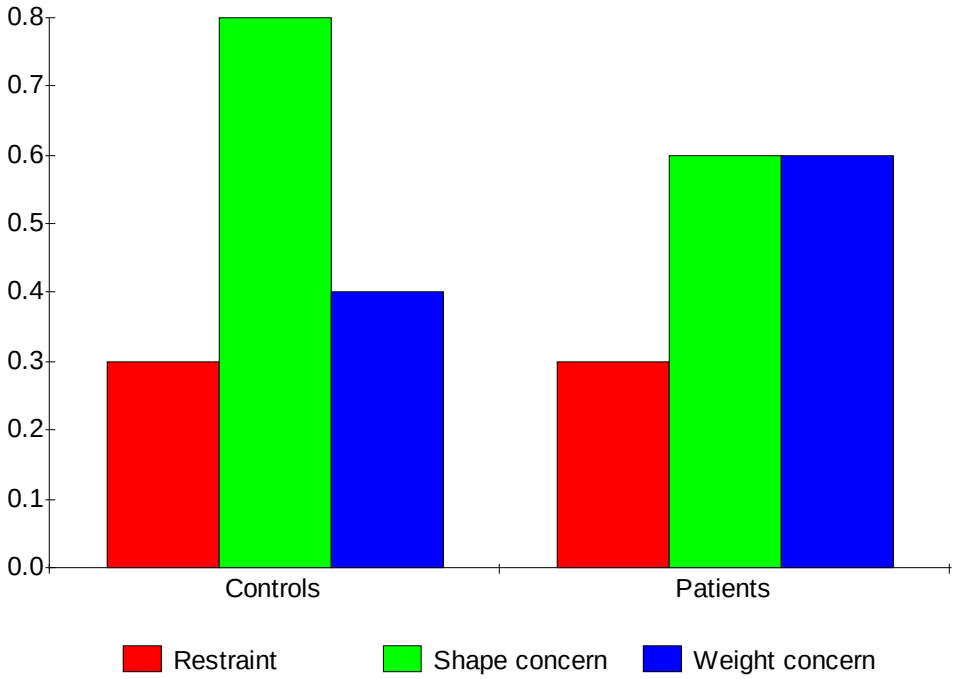
On the measurement of other behaviours (restricting food, and concern about shape and weight), the EDE-Q

[14] Order effects are confounding factors produced by the first condition that affect the second condition, like tiredness or boredom the second time. They cannot be removed, but counterbalancing evens them up. So if all individuals are bored the second time, this is evened up by half the participants doing interview then self-report, and the other half doing self-report then interview.

[15] Excessive use of laxatives to remove food after eating.

[16] A significant difference between the scores showed that individuals were reporting different information on the two measures. This research actually would like non-significant differences.

scores were always higher in both samples, and significantly different in most cases (figure 42.1).



(All significant differences between two measures at p<0.001 except weight concern for patients (p<0.01) and restraint for patients (not significant).

Figure 42.1 - Mean of differences between two measures (EDE-Q rating minus EDE rating).

Both EDE measures use similar wording, the same rating scale, and assess the same features over the same period of time, so any differences between them must be due to the mode of administration. Overall, the researchers recommended self-report questionnaires for behaviours that are not ambiguous or pose a problem of definition.

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43. BINGE EATING DISORDER - A REAL DISORDER?

DSM-IV (APA 1994) included a number of "provisional" mental disorders for discussion and research with the possibility of full inclusion in the next DSM. Binge eating disorder (BED) is one such case.

The key symptom is recurrent binge eating without compensatory behaviour, like self-induced vomiting (as in bulimia nervosa) or extreme dieting (as in anorexia nervosa) (box 43.1).

1. Recurrent episodes of binge eating that occur in a discrete period of time (within two hours) and a sense of loss of control during the eating.

2. The binge episodes are associated with three of the following: eating
(*i*) more rapidly than normal,
(*ii*) until feeling uncomfortably full,
(*iii*) large amounts of food when not hungry,
(*iv*) alone due to embarrassment or guilt, and
(*v*) amounts that lead to feeling disgusted, depressed, or very guilty.

3. Marked distress regarding binge eating is present.

4. Binge eating occurs at least two days a week for a six-month period.

5. The binge eating is not associated with the regular use of inappropriate compensatory behaviours such as purging, fasting, or excessive exercise and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa.

(Source: Striegel-Moore and Franko 2008)

Box 43.1 - Diagnostic criteria for BED in DSM-IV.

Blashfield et al (1990) proposed five criteria to use when deciding to make a provisional mental disorder into a "full" one:

i) There is ample research on it;
ii) The diagnostic criteria are clear and assessments instruments exist for them;
iii) Inter-rater reliability in diagnosis (ie: two different individuals give the same diagnosis based on the symptoms);
iv) Evidence that the condition is distinct from similar ones;
v) Validity of the condition.

Striegel-Moore and Franko (2008) used these criteria

to argue that BED should be included in DSM-V as a full mental disorder.

1. Ample research.

Striegel-Moore and Franko (2008) reported over 1000 articles on BED in a PubMed database [17] search in 2007. I put the term "binge eating disorder" into Google Scholar (http://scholar.google.com/) (on 12/11/09), and 1170 hits were reported in the title and 9790 "anywhere in the article".

But quantity of academic interest does not mean that a condition is "real". Using Google Scholar again, there were 2520 hits for "astrology" in the title of a reference.

2. Clear, measurable diagnostic criteria.

Box 43.1 shows the diagnostic criteria for BED, which on the face of it seem clear. But how to operationalise the criteria? For example, "large amounts of food" and "marked distress". It seems obvious, but clearly unambiguous, measurable terms are needed. Also the time periods mentioned for diagnosis are important (two days a week).

Striegel-Moore and Franko (2008) felt that measuring instruments, like the Eating Disorder Examination (EDE) (Fairburn and Cooper 1993), were usable - showing reliability and discrimination for BED.

3. Inter-rater reliability in diagnosis.

The ideal for inter-rater (or inter-clinician or inter-judge) reliability should be at least 70% agreement (correlation of 0.7 or better). Striegel-Moore and Franko (2008) reported finding no studies testing for this with BED. But there were studies showing test-retest reliability. This is diagnosis by the same clinician at two different points in time (eg: Stice et al 2000: one-week test-retest reliability of 0.75).

4. Distinct from other conditions.

There is overlap in symptoms between disorders, but there must be enough differentiation between each one. In

[17] A database of medical and related academic articles maintained by the US National Library of Medicine (http://www.ncbi.nlm.nih.gov/pubmed).

this case, BED overlaps with bulimia in terms of recurrent binge eating behaviour. Bulimia is different in that sufferers compensate for the binge with purge behaviour.

There are various ways of differentiating similar disorders:

- Demographics of sufferers - Bulimia is predominantly among White girls and young women, while BED sufferers are also older and other ethnic groups (Striegel-Moore and Franko 2008).

- Nature of symptoms - eg: difference in types of food binged.

- Course of disorder - ie: how it develops in terms of improvements and declines, and length of illness.

- Outcome and response to treatment.

- Co-morbidity with other disorders.

5. Validity of the condition.

Generally, validity refers in research methodology to an assessment instrument that measures what it claims to measure. For example, size of skull among humans is not a valid measure of intelligence, though it was used in the nineteenth century.

In terms of mental disorders, one way to establish validity is through the presence of symptoms together. If one symptom is exhibited, there should be at least a 50% chance that another symptom is present (Blashfield et al 1990). Striegel-Moore and Franko (2008) reported no studies for BED with sufferers, but there were statistical approaches to symptom co-existence (eg: latent class analysis).

Other types of validity include (Brewer 2001):

- Aetiological validity - Historical antecedents of the condition (eg: cause is same for all sufferers).
- Concurrent validity - Common characteristics among all sufferers.
- Predictive validity - Prediction of outcome of the illness from diagnosis.

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44. THE POSITIVE EXPERIENCE OF ANOREXIA

Warin (2004) noted, from her study of "women living with anorexia", that sufferers can "desire" the condition because it is an "empowering state of destruction", at least in part. This make anorexia different to other conditions that harm the individual's health, as shown by the development of "pro-anorexia" ("pro-ana") websites that encourage individuals to become anorexic. Such websites are "illustrative of the ways in which for many people, particularly young women, anorexia is central to the formation of particularly subjectivities around which relationships may be formed with the self, and others" (Rich 2006 p285). As of yet, there tends not to be equivalent pro websites for cancer, arthritis, and other chronic medical conditions.

Anorexia is viewed negatively in society, and thus sufferers are stigmatised. But sufferers have to live with this and the condition, and to make sense of their identity. One way is to gain support from other sufferers in "the secret language of eating disorder" (Warin 2004).

VIRTUAL SUPPORT

Giles (2006) analysed twenty pro-ana websites over two periods in 2003. "Discussion forums" and "message boards" were studied using a technique from conversation analysis called "membership categorisation analysis" (Baker 2004). It explores "the ways in which speakers use category information and inferences to create a frame of reference to make meaning of the world" (Giles 2006 p467).

"Central categories", like ingroup and outgroup, that underpin talk are highlighted and how the speakers make use of those categories. For example, "haters" are the outgroup. These are individuals who post negative comments about eating disorders, and are not pro-ana.

The outgroup also includes "normals" (an individual without an eating disorder), and "fakers" and "wannabes" (individuals claiming to have an eating disorder but not seen as a member of the ingroup). Responding to the postings by members of the outgroup is a way the ingroup solidarity is maintained and enhanced.

"Haters" are a challenge to the safe haven of the pro-ana community, whereas "wannabes" are a threat to the "elitism" of "true anas": "You've actually gotta be ..quite good at ..controlling yourself to be anorexic. Most people can't manage it" (Respondent in Burns 2004 quoted in Giles 2006 p472).

The ingroup is also concerned about who should be a

member as shown in the "ana/mia debate" (anorexia/bulimia). "Ana" is presented as superior to "mia": "I always found something pure about ana, but mia I think would be easier.." (extract 4; Giles 2006 p468). Anorexia is presented as "a club with stringent entry criteria" (Giles 2006).

Giles (2006) concluded about the maintenance of community boundaries in pro-ana websites:

> If anorexia is about attempting the unachievable (forever chasing impossibly low weights, trying to live on air), then it is not surprising that anorexics set such high standards for admittance to their community. The pro-ana world then becomes a hierarchy of in-group (positive identities), with ana ("true" or "pure" ana to be precise) at the top, mia just below, and marginal, vague identities such as ED-NOS at the margins. Ironically, for a community that actively resists social norms, medical diagnosis operates as a qualification – but this only serves to point up a critical ambivalence towards medicine in the pro-ana community, where site users debate endlessly whether ana is an "illness" or a "choice". Diagnosis serves as nothing more than a mark of authenticity (p475).

FACE-TO-FACE SUPPORT

Rich (2006) used interviews with young women (11-18 years old) at a treatment centre for eating disorders in the UK to see how sufferers made sense of their anorexia or bulimia. Using ethnography, the researcher also tried to access the "subculture" of anorexia. A number of themes emerged from the data collected:

i) The stigmatisation of anorexia - The women reported feeling misunderstood, and even alienated from others because of the pathology of their behaviour and its consequent stigmatisation. There was a feeling that others focused on the weight lost (or gained) and eating "rather than understanding the social and emotional dimensions of what it is like to be a young woman suffering from an eating disorder".

For example, one of the women reported a case where a teacher in the school dining room had said, "is this really you in a queue for food?". "It was awful, I mean, especially there in front of all those people" (Hayley; Rich 2006 p289).

Put simply, the focus on the body and the negative reactions to being so thin become internalised, and "these young women diagnosed with eating disorders came to be viewed as 'different' from their peers or 'deviant'" (p293).

ii) Reacting to the stigma - The reaction to such negative responses in society and others to anorexia was to find support with other sufferers. This showed itself in the sharing of "anorexic tricks" (Warin 2002; quoted in Rich 2006) (eg: pretending to eat food while really hiding it).

The sharing of practices and techniques was a way of constructing an identity as a "successful" anorexic which afforded status and empowerment [18]. Lauren noted: "I truly believed that my role in the world, my place, was to be the anorexic. Because you have the dominants, the leaders, the thinkers, I was just the anorexic, that was who I was" (Rich 2006 p298). later she said, "It [anorexia] shows that you have a strength that others don't, because, let's face it, not many people have the ability to starve themselves to death ..all the girls seemed to, I don't know, idolise it" (p298).

Hayley, in a similar vein, admitted: "I always used to look at my friends and think that I wanted to be as good, or as pretty, or as clever as them. So I decided that not eating was a way that I could maybe achieve that" (p299).

iii) Anorexic disconnection - The young women experienced the anorexia as contradictory - as positive but also aware of its destructive side. This related most prominently to the health effects, but also to the relationship with other sufferers - as supportive and competitive; eg: "And when this other girl at the school became anorexic, I felt I had been pushed out of my place and I was furious" (Lauren; Rich 2006 p300).

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[18] In terms of identity generally, Nichter (2000) observed how expressing the desire to lose weight (even if not actually doing it) was a way of "group belonging" among teenagers in Tucson, USA.

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45. QUESTIONNAIRE RESEARCH ON ATTACHMENT, CHILDHOOD, AND EATING DISORDERS

From a psychoanalytic point of view [19], eating disorders can be seen as an externalisation of issues from a lack of secure attachment in the early years. The "unconscious desire" to have the attachment, usually with the mother, that was denied is manifest in controlling the body. Put simply, "if I am thin, I will be lovable". The upshot is that sufferers of eating disorders will have insecure attachments compared to non-sufferers.

For example, Sharpe et al (1998) found a relationship between insecure attachment, and weight concerns, and low self-esteem among pre-adolescent girls and adolescents. But there are two methodological problems with establishing such relationships (Troisi et al 2005):

- How to measure attachment;
- Whether to measure attachment to significant others now or in the early years.

Troisi et al (2005) were conscious of these issues in their study in Rome, Italy of 78 women with eating disorders and sixty-four healthy female controls. The latter group were recruited from students attending paramedic courses and young women hired to applaud at a television show.

In relation to the two problems mentioned above:

i) Attachment was measured by the Attachment Style Questionnaire (ASQ) (Feeney et al 1994) which includes forty statements rated on a six-point scale.

It measures confidence (in the self and others), discomfort with closeness, need for approval, preoccupation with relationships, and relationships as secondary to achievement. A low score on confidence is a sign of an insecure attachment as confidence is a characteristic of a secure attachment. The other characteristics are those of insecure attachment (high score). The focus was upon current attachment feelings (which are assumed to originate in childhood).

ii) Attachment in childhood specifically was measured by the Separation Anxiety Symptom Inventory (SASI) (Silove et al 1993) [20] which contains fifteen symptoms of separation anxiety rated between 0-3

[19] Psychoanalysis (based on the ideas of Sigmund Freud) focuses upon the early experiences (first few years of life) and the unconscious mind as crucial in explaining adult behaviour.

[20] Based questionnaires were Italian versions (Troisi et al 2001).

(frequency in childhood). A higher score means greater separation anxiety as a child.

Overall, there was a relationship between SASI score (amount of separation anxiety symptoms reported) and ASQ score (secure or insecure attachment), which was especially strong for women with anorexia. The mean SASI score was 11.48 for controls and 16.62 for anorexics (and 15.85 for bulimics). This study confirmed a link between insecure attachment (in the past and now) with eating disorders, though it was dependent upon recall accuracy with the SASI.

ATTACHMENT TO FATHERS

Most of the work on attachment concentrates on the mother-child relationship. However, the father-daughter relationship has been found to play a role for women with eating disorders. For example, women with bulimia can have fathers who give insufficient care and high overprotection, or are rejecting, withdrawn or passive fathers (Jones et al 2006).

Jones et al (2006) were interested in how the father-daughter relationship had an influence on eating disorder development through the core beliefs that women held about themselves. Sixty-six women with eating disorders from the Eating Disorder Association (EDA) and fifty female student controls completed three questionnaires:

i) Eating Disorder Inventory (EDI) (Garner et al 1983) - measures severity and type of eating disorder;

ii) Young Schema Questionnaire - Short Form (YSQ-S) (Young 1998) - measures fifteen core beliefs with 75 statements (table 45.1);

iii) Egna Minnen Betfaffande Uppfostran (My memories of upbringing) (s-EBMU) (English version) (Arrindell et al 1999) - measures memories of parents' child-rearing behaviour in terms of rejection, emotional warmth, and protection. it is completed separately for each parent, but only scores for father were analysed.

It was hypothesised that parents' child-rearing behaviour would predict core beliefs which would predict eating disorders (figure 45.1).

CORE BELIEF	DESCRIPTION
Abandonment	Feeling that close relationships will end imminently
Dependence/incompetence	Feeling that cannot cope without others
Defectiveness/shame	Feeling of being internally flawed and unlovable
Emotional dependence	Feeling that emotional needs will never be met
Emotional inhibition	Belief that emotions must be inhibited
Enmeshment	Emotional over-involvement with others
Entitlement	Feeling that cannot act without consideration of others
Failure to achieve	Belief that incapable of performing well
Insufficient self control/self discipline	Feeling that impulses and feelings cannot be controlled
Mistrust/abuse	Feeling that others will hurt or use you
Subjugation	Belief that must submit to desires of others
Social isolation	Feeling of being different and isolated from others
Self-sacrifice	Belief that others' needs must be satisfied
Unrelenting standards	Belief in striving for high standards
Vulnerability to harm and illness	Feeling of no control over threat of disaster

(After Jones et al 2006)

Table 45.1 - Core beliefs measured by YSQ-S.

PARENTS' CHILD-REARING BEHAVIOUR - father	→	CORE BELIEFS ABOUT SELF, RELATIONSHIPS, AND WORLD	→	PRESENCE OF EATING DISORDER
(s-EMBU score)		(YSQ-S score)		(EDI score)
		THUS		
Negative parental child-rearing behaviour	→	Unhealthy beliefs	→	Eating disorder
High rejection/ protection scores; low emotional warmth		High score		High score
		OR		
Positive parental child-rearing behaviour	→	Healthy beliefs	→	No eating disorder
Low rejection/ protection scores; high emotional warmth		Low score		Low score

Figure 45.1 - Relationship predicted between three questionnaires.

The results supported the predicted relationship between the questionnaires. Eating-disordered women had significantly higher scores on the s-EMBU and the YSQ-S (figure 45.2).

s-EMBU	→	YSD-S	→	EDI
Higher on rejection and protection, lower on emotional warmth	→	Higher on all 15 core beliefs	→	Significantly higher scores

Figure 45.2 - Results of questionnaires for women with eating disorders.

More detailed analysis found that high paternal rejection (s-EMBU) predicted high defectiveness/shame and abandonment beliefs (YSQ-S) which predicted a drive for thinness and body dissatisfaction (EDI). The other key belief was found to be vulnerability to harm.

LATER CHILDHOOD

Attachment research is focused upon the early years

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of life, but the relationship and behaviour of parents throughout childhood plays a role. For example, the attitudes and behaviour of mothers towards food during the child's life.

Stein et al (2006) investigated the attitudes towards food of ten-year-olds of thirty-three mothers with a history of eating disorders during the children's first year of life (index group) and 23 controls. The children's attitudes were assessed on the child version of the Eating Disorder Examination (Child - EDE) (Bryant-Waugh et al 1996).

The children of the index group scored significantly higher of the Child-EDE than controls, though none were diagnosed with an actual eating disorder. "Thus children in the index group were more likely to be exhibiting dietary restraint and to hold overvalued ideas about weight/shape in their schema of self-evaluation" (Stein et al 2006 p327). This could be a precipitating factor for future development of eating disorders.

The length of the mother's eating disorder was a crucial variable, as was mother -child mealtime conflict from age five years.

PSYCHOMETRIC QUESTIONNAIRES

The behaviours and attitudes in these studies were measured by using psychometric questionnaires. These are questionnaires that have been designed and standardised, and assessed for reliability, validity and discrimination (table 45.2). They have a number of strengths and weaknesses (table 45.3).

CRITERION
RELIABILITY - consistent both within itself (internal reliability) and over time (external reliability). Internal reliability established by the split-half method, Kuder-Richardson method, or Cronbach's alpha. External reliability by test-retest or parallel forms method.
VALIDITY - measure the behaviour that it claims to measure. The main types of validity are face, content, criterion (made up of concurrent, and predictive validity), and construct validity.
STANDARDISATION - when the test is first constructed, it must be adjusted to approximate to a normal distribution of scores. Most tests have norms ("normal scores") for the majority of the population.
DISCRIMINATION - individual items and the test as a whole discriminate between high and low scorers; eg: individual with severe depression should answer items different to individual not depressed. Item analysis is the main technique used here.

Table 45.2 - Criteria for good psychometric test.

STRENGTHS	WEAKNESSES
1. They have standardisation, discrimination, reliability and validity. 2. An individual's data can be compared to other individuals. 3. An individual's data can be compared to norms. 4. Allows comparison between studies using same psychometric questionnaires.	1. Depends on honesty and accuracy of recall of respondents. 2. Depends on accuracy of construction and standardised sample used. 3. Decisions for cut-off points are often subjective. 4. Assumes that items and statements are measuring something real underlying the symptoms.

Table 45.3 - Strengths and weaknesses of psychometric questionnaires.

Psychometrics as a subject area concentrates on techniques for measuring psychological processes, and the technique can dictate the way the world appears - "When the only tool you have is a hammer everything begins to look like a nail" (Richards 2002).

Furthermore, Butt (2007) observed that: "We too readily think that knowledge is discovered, but it is constructed as well as discovered - what we think of as facts are made as well as found. Patterns of behaviour do not lie around waiting to be picked up, they are only viable within the searchlight of a particular theory" (p189).

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46. THE CREATION OF BODY DISSATISFACTION AMONG WOMEN

The continual presentation of images of thin women in the Western media has produced a situation where most women are dissatisfied with their bodies. Rodin et al (1984) called this, "normative discontent".

This is worrying because body dissatisfaction is linked to the development of eating disorders. While, from a feminist viewpoint, the focus upon the body "can steal resources (eg: time, attention, monetary resources) from other issues and activities that might empower women, rather than making them feel inadequate" (Engeln-Maddox 2005).

The myriad of media images of thin women (between 10-20% below expected weight; Wiseman et al 1992) links such physiques to physical attractiveness. This is internalised as social rewards for physical attractiveness among women, emphasised in society, and manifest as body dissatisfaction. Social comparison plays a role as individuals evaluate themselves in relation to others. Media figures are ideals and possess "expertise", and thus become models for comparison. "Because few women can meet the beauty standards created by this ideal, dissatisfaction with one's own appearance is a likely outcome of this comparison process" (Engeln-Maddox 2005).

Studies using different methods have shown the link between exposure to idealised media images of thin women and body dissatisfaction.

i) Correlational studies - eg: Harrison and Cantor (1997).

232 female and 190 male undergraduates in communication courses at the University of Wisconsin-Madison, USA were asked to complete surveys about their media exposure, and attitudes towards eating and dieting. These were the two variables for the correlation.

- Media exposure - eg: number of hours of television watched; frequency of viewing certain popular television shows with thin bodies (eg: "Melrose Place") and heavy bodies (eg: "Roseanne").

- Attitudes towards eating and dieting - eg: "Am preoccupied with desire to be thinner", "I think my hips are too big", "I have gone on eating binges where I felt that I could not stop".

For women, media exposure was significantly correlated with disordered eating for magazines read per

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week, but not for overall television viewing. There was not a correlation between type of television show (eg: thin bodies) and disordered eating. But overall television viewing did correlate with body dissatisfaction.

For men, media exposure correlated with positive attitudes towards perceived personal thinness and dieting, and with a favourable attitude towards it for women.

ii) Quasi-experiments - eg: Becker et al (2002).

Becker et al looked at the effect on Fijian adolescent females of the arrival of television to the Nadroga province of Fuji [21]. Data were collected from two separate groups in 1995 (soon after television arrived) and 1998 using the EAT-26 questionnaire (Garner 1982) (in English - the language of education - and the local Fijian dialect). Scores above twenty are treated as a sign of an eating disorder.

In the 1998 sample significantly more adolescents had an EAT-26 score greater than twenty (29.2% vs 12.7%), and reported self-induced vomiting to control weight (11.3% vs 0%) than in 1995.

Table 46.1 summarises the limitations of this study, and table 46.2 outlines controls that a "true" experiment would have. However, performing such a controlled experiment is very difficult in social situations.

1. As a quasi-experiment variables were not manipulated by the researchers and full randomisation to groups did not take place. Thus it is necessary to be cautious about cause and effect compared to a laboratory experiment.

2. No baseline before television arrived. Quasi-experiments tend to be opportunist studies that make use of a social event - in this case, the arrival of television to parts of Fiji.

3. No details about samples before 1995, and some of these individuals may have had disordered eating before television arrived.

4. Eating disordered behaviour was based on self-reports and not clinical diagnosis by psychiatrists.

5. Small sample sizes (n = 63 in 1995, n = 65 in 1998).

6. No investigation of other social variables than television that could explain increase in eating disordered behaviour.

Table 46.1 - Limitations of Becker et al (2003) study.

[21] See map at http://tinyurl.com/yeqw94m.

- Baseline measure of behaviour - before television arrived.
- Details of participants at baseline - eg: how many already showed eating disordered behaviour.
- Random allocation into two samples.
- Measurement of other relevant social variables during the study.
- Ideally, interview same individuals at two points in time (ie: longitudinal study).

Table 46.2 - Controls needed for experiment rather than quasi-experiment.

iii) Experiments - Groesz et al's (2002) meta-analysis of twenty-five experiments that manipulated the images presented found that body image was significantly more negative after viewing thin models compared to average size models, plus size models, or inanimate objects.

COUNTERING THE IMAGES

Yet women are not passive consumers, they can be critical in processing these images. However, there is a paradox "that while women can be hurt by these images, they are often quite astute when it comes to critically processing media messages" (Engeln-Maddox 2005).

Studies have looked at "interventions" to encourage critical processing of the media. For example, Posavac et al (2001) used an educational programme, including information about the real body shape of women, before showing women with high levels of body dissatisfaction images of thin women. This group were less likely to report weight concerns or make negative self-evaluations afterwards compared to the control group.

But not all women respond to such interventions nor do all women develop eating disorders. So there must be individual factors involved in both cases.

Engeln-Maddox (2005) investigated factors in body dissatisfaction among 202 female college students in the psychology department of a private university in the USA. The participants were randomly divided into Phase A or Phase B of the study first (ie: counterbalanced).

Phase A

Ninety-two women were presented first with sets of adverts from women's magazines showing faces, bodies, or distractors (ie; non-physical images). Participants were asked to list the first ten thoughts they had in response to the adverts. The thoughts were coded into five categories - counter-arguments, negative outcome social comparisons, positive outcome social comparisons,

positive assessment of model's appearance, and other (table 46.3). 110 women performed phase A second.

CATEGORY	DESCRIPTION
Counter-arguments	Criticism of model for being too thin, too perfect, fake or airbrushed
Negative outcome social comparisons	Expression of dissatisfaction with own appearance
Positive outcome social comparison	Positive comment about own appearance
Positive assessment of model's appearance	Positive comment about model's physical appearance
Other	Comment about product of advert

Table 46.3 - Description of coding categories used.

Phase B

One hundred and ten women did this phase first and 92 women did it after phase A. This phase of the study involved completion of various questionnaires about attitudes towards the body.

- Sociocultural Attitudes Towards Appearance Questionnaire - Internalising Scale (SATAQ-I) (Heinberg et al 1995) - eight statements measuring the degree of internalisation of the thin ideal.

- Multidimensional Body-Self Relations Questionnaire - Appearance Scales (MBSRQ-AS) (Cash 2000) - measures the importance that an individual places on their appearance.

- Eating Disorder Inventory-2 (EDI-2) (Garner 1991) - the body dissatisfaction subscale was used from this measure of eating disorders.

- Satisfaction With Life Scale (SWLS) (Diener et al 1985) - five statements about overall satisfaction with life.

Means for the whole group were calculated for the questionnaires (figure 46.1). satisfaction with life was above the mid-point as was internalisation of the thin ideal, and importance of appearance, but body dissatisfaction was below the mid-point. Internal reliability was calculated for each questionnaire using Cronbach's alpha (appendix 46A).

MINIMUM			MAXIMUM
	SWLS		
5		**23.24**	35
low satisfaction with life			high
	SATAQ-I		
8		**25.34**	40
low internalisation of thin ideal			high
	EDI-2		
0	**9.03**		27
low dissatisfaction with body			high
	MBSRQ-AS		
1		**3.40**	5
low importance of appearance			high

Figure 46.1 - Mean scores on questionnaires

The scores on the different questionnaires were then correlated. As expected, internalisation of thin ideal (SATQ-I score) significantly positive correlated with body dissatisfaction (EDI-2 score) and importance of appearance (MBSRQ-AS score). In other words, high scores on all three of these questionnaires went together as did low scores.

Overall, making negative outcome social comparison thoughts in response to pictures of women (phase A) was associated with greater internalisation of the thin ideal and greater body dissatisfaction. This is a correlational study, so the direction of the relationship between these variables cannot be established. For example, the behaviour in phase A could predict the behaviour in phase B or vice versa.

APPENDIX 46A - Cronbach's alpha

Internal reliability refers to the consistency of an individual respondent in answering the questions. This can be established by correlating the answers on even number questions with odd number questions, for example (split-halves correlation). Cronbach's alpha (Cronbach 1951) is a more sophisticated calculation of this, and the closer the number to one the better for internal reliability. Table 46.4 gives the Cronbach's alpha scores

for the questionnaire used by Engeln-Maddox (2005).

QUESTIONNAIRE	CRONBACH'S ALPHA
SATAQ-I	0.81
MBSRQ-AS	0.84-0.87
EDI-2	0.87
SWLS	0.88

Table 46.4 - Cronbach's alpha scores in Engeln-Maddox (2005) study.

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47. CELEBRITY WORSHIP AND BODY IMAGE DISTURBANCE

It is probably too simple to suggest that mere exposure to media images of thin models leads directly to eating disorders. There must be other stages or mechanisms involved like body image disturbance. One such factor is the "para-social relationship" (Giles 2002) (appendix 47A) with media figures, particularly of adolescents, manifest as "celebrity worship" [22] (figure 47.1).

Heilman (1998) described a fourteen year-old called "Kara" who wanted to be like the fashion model, Kate Moss. She dieted to emulate the Moss figure and was eventually diagnosed as suffering from anorexia nervosa.

MEDIA IMAGES OF THIN MODELS	→	CELEBRITY WORSHIP	→	OWN BODY DISSATISFACTION/ BODY IMAGE DISTURBANCE	→	EATING DISORDERS

Figure 47.1 - A theoretical link between media images and eating disorders.

McCutcheon et al (2002) explained celebrity worship through an "Absorption-Addiction" model. Adolescents, in particular, with identity problems become psychologically absorbed with a celebrity as a way to establish identity. The positive feelings associated with the celebrity become addictive and produce more focus on the celebrity. The degree to which this happens depends upon the level of celebrity worship.

Celebrity worship has three increasing levels of involvement (Maltby et al 2005):

i) Low level for "Entertainment-social value" - interest in the celebrity relates to the entertainment value of that famous person;

ii) Intermediate level for "Intense-personal feelings" - signs of obsession about the celebrity;

iii) Extreme level labelled "Borderline-pathological" - the celebrity is the centre of the individual's life to the exclusion of many other things.

[22] "A celebrity is 'known for being well-known' ..., regardless of whether that eminence derives from the entertainment field, medicine, science, politics, religion, sports, or close association with other celebrities" (McCutcheon et al 2002 p67).

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The level of celebrity worship has been linked to the personality theory of Hans Eysenck (Eysenck and Eysenck 1985): Entertainment-social/Extraversion, Intense-personal/Neurotic, and Borderline-pathological/Psychoticism (Maltby et al 2003). While Maltby et al (2006) found correlations between Intense-personal and fantasy proneness [23], and Borderline-pathological and fantasy proneness and dissociation [24].

Maltby et al (2005) predicted that Intense-personal feelings about a celebrity would be linked to body image concerns, particularly for female adolescents and same-sex celebrities. Three samples of participants from the north of England were used for this research:

- 229 adolescents (14-16 years) (127 female/102 male);
- 183 university undergraduates (95 female/88 male);
- 289 adults from the general population (163 female/126 male).

All participants were asked to complete three questionnaires:

- Celebrity Attitude Scale (CAS) (McCutcheon et al 2002) (appendix 47B) - 22 statements relating to the different aspects of celebrity worship; eg: "learning the life story of my favourite celebrity is a lot of fun" (Entertainment-social), "I consider my favourite celebrity to be my soul mate" (Intense-personal), and "if I were lucky enough to meet my favourite celebrity, and he/she asked me to do something illegal as a favour I would probably do it" (Borderline-pathological). Each statement has a five-point response choice. Scores range from 23 to 115.

- Attention to Body Shape (ABS) (Beebe 1995) - Seven items rated on a five-point scale about the importance of body shape and weight to the individual. Range of scores from 7 to 35.

- Body Shape Questionnaire - Revised (BSQ-R) (Mazzeo 1999) - Ten statements rated on a six-point scale about preoccupation with body image. Minimum score is ten and maximum 60.

The adolescent sample had higher mean scores on the

[23] Measured by agreement to statements like "Many of my fantasies have a realistic intensity" on Creative Experiences Questionnaire (CEQ) (Merckelbach et al 2001).

[24] Measured by the Dissociative Experiences Scale (DES) (Bernstein and Putman 1986) with statements like "Some people have the experience of looking in the mirror and not recognising themselves".

CAS showing a greater preoccupation with celebrities than other age groups (table 47.1). All age group females scored higher than males on the two questionnaires about body shape and image (figure 47.2).

AGE GROUP	MALES	FEMALES
ADOLESCENTS	52.22	55.69
STUDENTS	49.20	48.57
ADULTS	40.99	41.40

Table 47.1- Mean total scores on CAS.

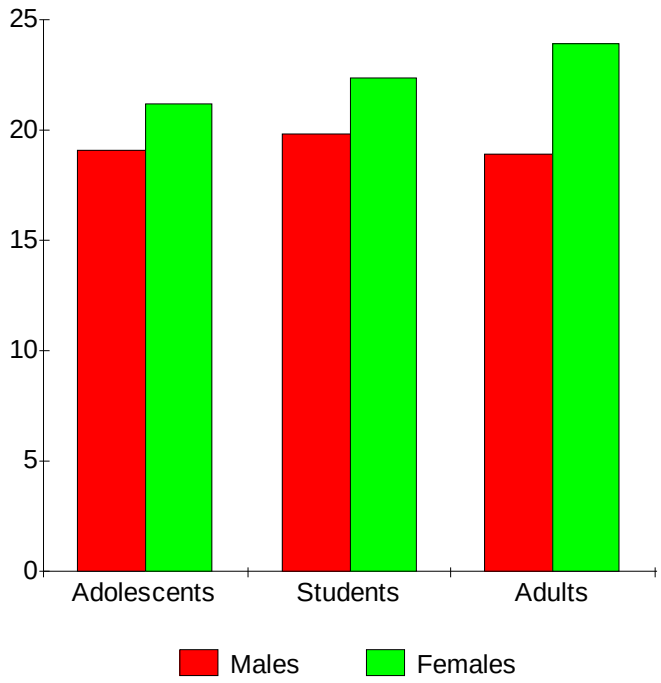


Figure 47.2 - Mean scores on ABS.

In terms of the relationship between CAS score and the other two questionnaires, only female adolescents showed a significant relationship. Importantly, a significant positive correlation of 0.42 was found for Intense-personal items of the CAS and the combination score on the questionnaires about the body ("Attention to Body Factor" score) using the Pearson product-moment correlation coefficient (table 47.2). When each year was analysed separately, the strongest relationship was at fifteen years old.

A significant positive correlation was also found among adolescents for the Borderline-pathological items, but the authors saw this as "the result of over-

enthusiasm or a tendency to make outrageous claims about the loyalty to a celebrity" (p24).

AGE GROUP	MALES	FEMALES
ADOLESCENTS	0.16	0.42 *
STUDENTS	0.18	0.16
ADULTS	0.15	-0.07

(* $p<0.01$)

(After Maltby et al 2005)

Table 47.2 - Pearson product-moment correlation coefficient between CAS Intense-personal score and Attention to Body Factor score.

This study is correlational, so it is not possible to establish causality. Three possible explanations could account for the correlations (Maltby et al 2005):

i) The Intense-personal feelings towards thin celebrities causes the female adolescent to develop a poor body image;

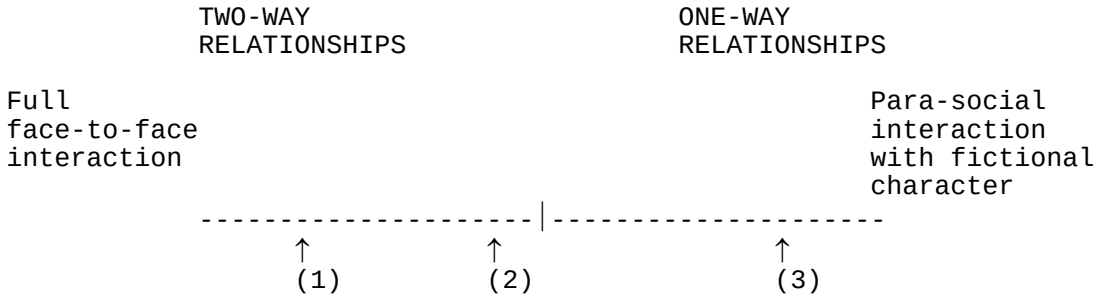
ii) Already having a poor body image causes the individual to focus upon a celebrity with an ideal body shape;

iii) Both the above working together.

APPENDIX 47A - PARA-SOCIAL RELATIONSHIPS

"Para-social relationship" (or para-social interaction; PSI) is a term coined to describe today "the interaction between users of mass media and representations of humans appearing in the media ('media figures', such as presenters, actors, and celebrities) ...to which the user responds as though in a typical social relationship" (Giles 2002 p279).

Giles (2002) suggested that PSIs with celebrities are part of a continuum of social interactions from full face-to-face reciprocity to PSI with a cartoon or fictional character. This idea can be presented as in figure 47.3, based on two-way or one-way interactions.



(1) Eg: interaction through electronic media, like chat-rooms.
(2) Eg: crush on older person at school who may or may not interact with.
(3) Eg: PSI with celebrity.

Figure 47.3 - Continuum of social and para-social interactions.

Importantly, PSIs are not new to celebrities in the media today. Such relationships existed in the past between citizens and leaders, for example (Caughey 1984).

PSIs show similar psychological processes to ordinary social interactions. For example, Gleich (1996; quoted in Giles 2002) asked individuals to rate friends, neighbours, and favourite media figures on a series of relationship dimensions. Friends were rated highest on these dimensions, but favourite media figures were rated more highly than neighbours.

APPENDIX 47B - CAS

1. If I were to meet this my favourite celebrity in person, he/she would already somehow know that I am his/her biggest fan.

2. I share with my favourite celebrity a special bond that cannot be described in words.

3. I am obsessed by details of my favourite celebrity’s life.

4. My friends and I like to discuss what my favourite celebrity has done.

5. When something good happens to my favourite celebrity I feel like it happened to me.

6. One of the main reasons I maintain an interest in my favourite celebrity is that doing so gives me a temporary escape from life’s problems.

7. I have pictures and/or souvenirs of my favourite celebrity which I always keep in exactly the same place.

8. The successes of my favourite celebrity are my successes also.

9. I enjoy watching, reading, or listening to my favourite celebrity because it means a good time.

10. I consider my favourite celebrity to be my soul mate.

11. I have frequent thoughts about my favourite celebrity, even when I don't want to.

12. When my favourite celebrity dies (or died) I will feel (or I felt) like dying too.

13. I love to talk with others who admire my favourite celebrity.

14. When something bad happens to my favourite celebrity I feel like it happened to me.

15. Learning the life story of my favourite celebrity is a lot of fun.

16. I often feel compelled to learn the personal habits of my favourite celebrity.

17. If I was lucky enough to meet my favourite celebrity, and he/she asked me to do something illegal as a favour, I would probably do it.

18. It is enjoyable just to be with others who like my favourite celebrity.

19. When my favourite celebrity fails or loses at something I feel like a failure myself.

20. If someone gave me several thousand dollars to do with as I please, I would consider spending it on a personal possession (like a napkin or paper plate) once used by my favourite celebrity.

21. I like watching and hearing about my favourite celebrity when I am in a large group of people.

22. Keeping up with news about my favourite celebrity is an entertaining.

(Source: Maltby et al 2005)

Entertainment-social - 10 items: nos 4, 6, 9, 13, 14, 15, 18, 19, 21, 22.

Intense-personal - 9 items: nos 1, 2, 3, 5, 7, 8, 10, 11, 12.

Borderline-pathological - 3 items: nos 16, 17, 20.

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48. LESBIAN, GAY AND BISEXUAL INDIVIDUALS AND EATING DISORDERS

There are different figures for how many individuals suffer from eating disorders, and these figures depend upon the definition of eating disorders and the method of collecting the data. However, disproportionately more women than men are sufferers, and usually they are younger individuals. Among male sufferers, disproportionately more are gay and bisexual men (eg: ten times more; Strong et al 2000).

One explanation for this "suggested that gay and bisexual men are more likely than heterosexual men to view their bodies as sexual objects, and therefore, like heterosexual women, may be more vulnerable to experiencing body dissatisfaction" (Feldman and Meyer 2007). On the other hand, by this explanation, lesbian and bisexual women are less prone to eating disorders "because they do not share with heterosexual women the standards of feminine beauty espoused by Western culture" (Feldman and Meyer 2007).

Feldman and Meyer (2007) set out to investigate the prevalence of eating disorders among lesbian, gay and bisexual (LGB) individuals by designing a study that overcame the weaknesses of previous ones.

i) Studies use imprecise terms and definitions like body dissatisfaction. For example, Williamson and Hartley (1998) found significantly higher body dissatisfaction scores among 41 young gay men compared to 47 heterosexual men in England.

Feldman and Meyer used DSM-IV diagnostic criteria for eating disorders. The World Mental Health Composite International Diagnostic Interview (WMH-CIDI), a fully structured measure, was used. This meant that the interviews lasted between 3-4 hours, and the participants were paid $80 for their time.

ii) Studies use convenience samples, like college students or those receiving treatment for eating disorders [25], which are biased.

Feldman and Meyer recruited respondents from "venues that catered primarily to LGB individuals" in New York City and the equivalent for the White straight sample (total of 274 venues visited between February 2004 and January 2005). Five hundred and sixteen individuals were

[25] Among a sample of thirty men and thirty women at the Maudsley Hospital, London for eating disorders, seven of the males (23%) were gay and only 3% (ie: 1) of the women (Bramon-Bosch et al 2000).

recruited (table 48.1).

However, the sample was still not a random one, and depended upon volunteers. Volunteers may over-represent individuals interested in the topic including suffering from an eating disorder. Furthermore, the study took place in one city in the USA.

The comparison sample of heterosexual individuals was quite small, and for convenience, only included White individuals.

LGB SAMPLE	HETEROSEXUAL SAMPLE
Total = 388 Gay/bisexual men = 193 • White = 65 • Black = 64 • Latino = 64 Lesbian/bisexual women = 195 • White = 67 • Black = 64 • Latino = 64	White men = 65 White women = 63

Table 48.1 - Ethnicity and gender of sample.

iii) Studies tend not to include ethnic differences in the LGB population. Feldman and Meyer deliberately recruited self-categorised White, Black/African American and Latino/Hispanic/Spanish members of the LGB population.

It was found that gay and bisexual men had the highest rate of any eating disorder (8.8%) compared to lesbian and bisexual women (7.2%), straight women (4.8%), and straight men (1.5%). Latino gay and bisexual men and women had the highest rates among ethnic groups (figure 48.1). Overall, gay and bisexual men were significantly more likely than straight men to suffer from eating disorders, while there was no difference between lesbian, bisexual and straight women.

The question is how generalisable are these findings:

- To all LGB individuals - NO: only in USA;
- To LGB individuals in the USA - NO: only in one city;
- To LGB individuals in New York City - NO: not random sample, but used volunteers.

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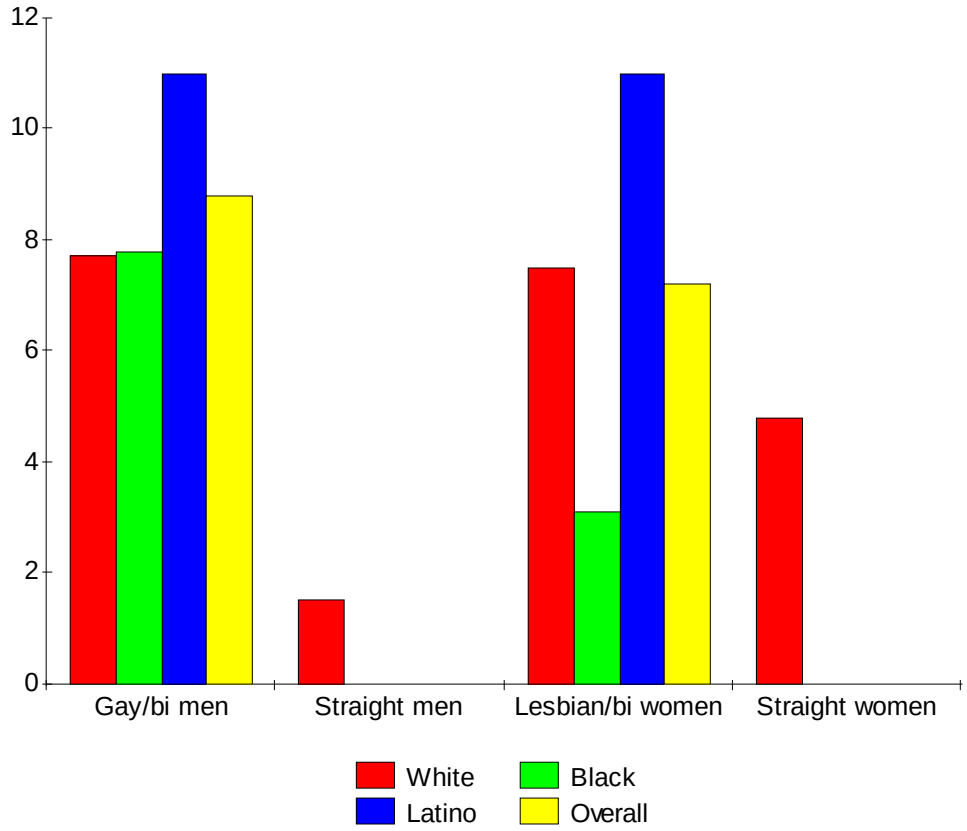

Figure 48.1 - Lifetime prevalence (%) of any eating disorder by ethnicity and sexual orientation.

However, the study did highlight important issues for practitioners. For example, it challenged the "commonly held conventions that lesbian and bisexual women are less vulnerable to eating disorders than heterosexual women, and similarly that racial/ethnic minorities are less vulnerable than whites" (Feldman and Meyer 2007).

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49. TWO MEASURES OF ATTITUDES TOWARDS THE BODY

Women in Western society are not happy with their body and its appearance, and the extreme version of this behaviour is seen in eating disorders like anorexia and bulimia. Researchers are, thus, interested in measuring attitudes towards the body. This article looks at the construction of two commonly used self-reported measures of concern about and focus on body shape.

Body Shape Questionnaire (BSQ) (Cooper et al 1987)

The first stage of development involved open-ended interviews about body shape, and, in particular, the experience of "feeling fat" with twenty-eight young women (most of whom had eating disorders). From the transcripts of these interviews, fifty-one questions were derived. These were presented as the draft questionnaire with a six-point response choice to four groups of women in Oxford and Cambridge (outpatients with bulimia, family planning clinic attendees, occupational therapy students, and university undergraduates).

Seventeen items were discarded because they did not discriminate between the groups. In other words, women with bulimia and students, for example, both chose the same or very similar replies. The final BSQ has thirty-four questions related to the past four weeks answered from "never" (1) to "always" (6) (minimum score = 34, maximum = 204) (table 49.1).

- Has thinking about your shape interfered with your ability to concentrate (eg: while watching television, reading, listening to conversations)?
- Has being naked, such as when **taking** a bath, made you feel fat?
- Have you avoided wearing clothes which make you particularly aware of the shape of your body?
- Have you imagined cutting off fleshy areas of your body?
- Has eating sweets, cakes, or other **high** calorie food made you feel fat?
- Have you not gone out to social occasions (eg: parties) because you have felt bad about your shape?
- Have you felt excessively large and rounded?
- Have you felt ashamed of your body?

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- Has worry about your shape made you diet?

- Have you felt happiest about your shape when your stomach has been empty (eg: in the morning)?

(Source: Cooper et al 1987 appendix)

Table 49.1 - Example of items from BSQ.

When these items were analysed based on the responses from the four groups of women, the means were significantly different. The bulimic women (n = 38) had a mean score of 136.9 and the other women together (healthy sample; n = 535) had a mean of 81.5. Discriminability was also shown by comparing two sub-groups of the healthy controls - one group who were currently dieting and one group not. The mean BSQ scores were 109.0 and 55.9 respectively.

The validity of the BSQ was then established. For concurrent validity, the scores of the BSQ were correlated with scores on similar measures of the same behaviour. As expected significant positive correlations were found between the BSQ and the Eating Attitudes Test (EAT) (Garner and Garfinkel 1979).

Attention to Body Shape (ABS) Scale (Beebe 1995)

Initially, nine statements were drawn up about focus on body shape from similar questionnaires. These were presented to twenty-two male and forty female undergraduates in an abnormal psychology class at a university in the USA on two separate occasions with a two-week gap. There was a five-point response choice. Two statements were removed because they were repetitious.

The final ABS (table 49.2) thus had seven items with response choices from "definitely disagree" (1) to "definitely agree" (5) (scores range from 7-35). Internal reliability (ie: correlation between items) was 0.70-0.82, and test-retest reliability of 0.76 for women and 0.87 for men.

1. I place a great deal of importance on my body shape.
2. I buy products that promise to give me a better body.
3. I am not self-conscious about my body shape.
4. I am always trying to improve my body shape.
5. I wear clothes that highlight the best aspects of my body and hide the worst aspects of my body.
6. It really bothers me when I can't keep my body in shape.
7. I'm very attentive to my body shape.

(Source: Beebe 1995)

Table 49.2 - Items of ABS scale.

Concurrent validity was established by comparison with other similar questionnaires using fifty-three psychology undergraduates. Construct validity is the correlation of scores on the test with an expected behaviour. In this case, it is expected that high ABS scorers would see and feel themselves to be larger and want to be smaller. Sixty-five female psychology undergraduates were asked to choose their body shape from a choice of nine figure drawings ranging from very thin to very fat, and then their ideal body shape. High ABS scorers had a larger discrepancy between their chosen shape and their ideal (ie: see themselves as larger and want to be smaller).

Table 49.3 summarises the details of the two measures.

	BSQ	ABS
Number of items	34	7
Response choices	6 point Likert scale	5 point Likert scale
Score range	34 - 204	7 - 35
Validation sample	Only female: bulimics and healthy controls	All university students, mostly female
Discrimination between different groups	Yes	Yes
Validity	Yes (Concurrent)	Yes (Concurrent, construct)
Reliability	No details given	Yes (Internal, test-retest)

Table 49.2 - Details of the two measures of attitudes towards the body.

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50. EATING DISORDERS AMONG ADULTS WITH LEARNING DISABILITIES: DIFFERENT TO GENERAL POPULATION?

In the two main diagnostic classification systems for mental disorders, DSM-IV (APA 1994) and ICD-10 (WHO 1992), most of the attention of eating disorders (EDs) is upon anorexia nervosa, bulimia nervosa, and binge eating disorder. There is "less interest in the atypical and less complex presentations of these three EDs: feeding relationship-based persistent feeding disorders, food faddiness/refusal and rumination/regurgitation; and other diagnosable EDs such as psychogenic loss of appetite, vomiting and overeating and pica.." (Gravestock 2003 p72). These are the conditions more likely among adults with learning disabilities (AWLD). ICD-10 and DSM-IV emphasis diagnosing anorexia and bulimia in young adults, and feeding disorders in children (Gravestock 2003).

But feeding disorders, abnormal eating behaviours (AEBs) and EDs are very common in AWLD (box 50.1) - up to 40% of institutionalised such adults and nearly 20% of community-living ones (Gravestock 2000).

- "Mervyn" (profound learning disabilities): food faddiness/refusal and rumination/regurgitation.
- "Paul" (severe LD and atypical autism): mixed pica (appendix 50A); eg: eating paper, stealing discarded food.
- "James" (mild LD and Down's Syndrome): binge eating disorder.
- "Christine" (mild LD and Prader-Willi Syndrome): constant eating as in binge eating disorder and food pica.

Box 50.1 - Examples of AEBs and EDs with different AWLD (Gravestock 2003) [26].

Concern that traditional diagnostic criteria are not entirely relevant to AWLD has led to the development of an alternative diagnostic system - "Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/Mental Retardation" (DC-LD) (RCP 2001) in the UK [27].

[26] Learning disabilities are usually categorised by IQ score; eg: mild (50-70), moderate (35-49), severe (20-34), profound (<20).

[27] The World Health Organisation produced the "ICD-10 Guide for Mental Retardation" (ICD-10-MR) (WHO 1996).

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There are a number of factors that can lead to differences in eating behaviour and EDs between AWLD and the general population.

i) Verbal abilities - Sufferers of anorexia in the general population, for example, usually can express verbally their thoughts and feelings about body image and emotions related to food in a way that many (non-verbal) AWLD could not.

ii) "Psychosocial masking" (Sovner 1986) - This is a term used to refer to restricted life experiences and opportunities that limit an individual's ability to show a particular behaviour. For example, bulimia sufferers having the opportunity to buy the food necessary for the binge, then eat it (usually alone) and purge afterwards, which institutionalised AWLD cannot do. So regurgitation by this latter group while being fed could be a version of the purging behaviour in bulimia.

iii) Different sufferer groups - For example, anorexia in the general population is associated with adolescent females, while younger male AWLD are vulnerable to pica and rumination/regurgitation (Gravestock 2003).

iv) Biological basis - While there is debate over the biological basis of EDs in the general population, with a condition like Prader-Willi Syndrome excessive eating is part of the disorder (ie: biologically impaired food satiety). Sufferers will continue to eat if food is available with no natural stopping when full. Damage to the hypothalamus seems to be involved (Holland et al 1995).

v) Co-morbidity - It may be that AEBs and EDs occur as co-morbidity to the learning disabilities like sleep problems, hyperactivity, and other behavioural problems.

Traditional classification systems have focused too much on anorexia, bulimia, and binge eating disorder such that there is a "probable underdiagnosis" of EDs in AWLD (Gravestock 2003). Thus the DC-LD attempts to rectify this narrow focus.

APPENDIX 50A - PICA

Pica is the compulsion to eat non-food items, and extreme pica occurs in 1-2% of AWLD (Saner 2006). Food pica involves eating food that is not prepared for eating (eg: coffee powder).

In extreme cases, sufferers risk poisoning, but more

often digestive problems. For example, a 61 year-old Welsh man, Dewi Evans, died in 2006 from an obstruction in the intestines. He had undergone surgery before to remove objects like screws, pen tops and coins from his bowel (Saner 2006).

In another case, an operation found five kilograms of coins and other small metal objects in the stomach of a 62 year-old French man who died soon after (Saner 2006).

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